DECEMBER

THE JOURNAL
OF THE
AMERICAN DENTAL ASSOCIATION
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Vice President of the American Dental Association

THE JOURNAL *of the*
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CLASSIFICATION AND HISTOPATHOLOGY OF PARODONTAL DISEASE

By KURT H. THOMA, D.M.D., and HENRY M. GOLDMAN, D.M.D., Boston, Mass.

THE etiology and pathology of parodontal diseases have been subjects of study by many men in the past. A review of the literature of the histologic aspect shows that there is as yet no consensus of opinion regarding the exact pathology of these diseases. The object of this paper is to record new evidence in the histopathology of parodontal diseases which was observed in the course of study of sixty-seven jaws obtained at necropsy.

PAST WORK

Talbot, working on material obtained from dogs, called attention to the inherent tendency of the supporting structures of the teeth to disappear. He described, as the earliest pathologic process, a marginal interstitial gingivitis producing round-cell infiltration in the marginal gingiva. He believed that this infiltration extended down through the capillary blood vessels into the submucous tissue as far as the periosteum of the bone, and that, on reaching there, the bone underwent the changes characteristic of halisteresis.

Znamensky found a deposit of tartar under the gingiva in all of his specimens. He considered the first pathologic change to be a dense infiltration with leukocytes occurring at the gingival margin, the bone remaining normal. The gingiva, he stated, became ulcerated later. He found that, when infiltration extended toward the bone, it became less dense as the bone was approached, and that when the bone was reached, it underwent transformation into osteoid and fibrous tissue.

Hopewell-Smith, in his studies, found that "pyorrhea" did not begin as a gingivitis, but was essentially dependent on an osseous lesion; namely, an atrophy of bone caused by halisteresis and osteoclasis. He believed that gingivitis was superimposed on the original condition.

Box, in his work, recognized two types of the disease. In his "simplex" type, he described a condition occurring at the base of the gingival crest, a round-cell infiltration, later followed by resorption of the alveolar crest. In his "complex"

(From the Laboratory of Oral Pathology, Harvard University Dental School.)

(Read at the Massachusetts State Dental Society, Boston, April 29, 1937.)

type, he described what he called "rarefying pericementitis fibrosis." This he interpreted as the "simplex" type superimposed on a noninfective atrophy of the tooth socket.

Gottlieb gave a clinical classification of parodontal diseases in which he distinguished between conditions developing as the result of inflammation of the gingival tissues and those developing from systemic disturbances. The first form he considered a result of malhygiene accompanied by inflammation, naming it "schmutz-pyorrhea" because of the uncleanliness and the accumulation of irritating débris and calculus in the gingival crevices. He found that the gingival tissues respond with hyperemia and engorgement, and finally breaks develop in the epithelium. The inflammation, gradually progressing down to the bone, causes inflammatory bone resorption. His second type, Gottlieb named "diffuse atrophy." He pointed out that early clinical symptoms of this type were manifestations of primary bone changes, resorption of bone as well as of root surfaces, with resultant widening of the peridental membrane and loosening of the teeth. He showed that, in this condition, pocket formation and suppuration were distinctly secondary symptoms.

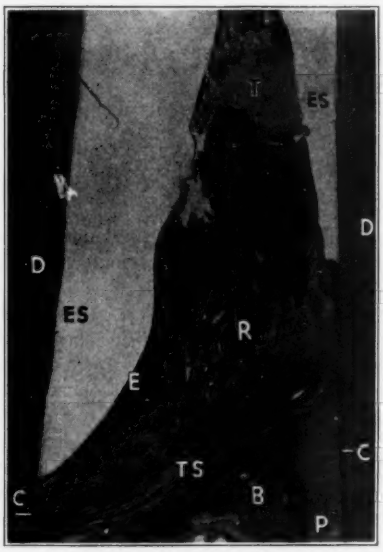

Fig. 1.—Gingivitis. D, dentin; T, débris; E, epithelium; R, round-cell infiltration; ES, enamel space; TS, transeptal fibers; B, bone; C, cementum.

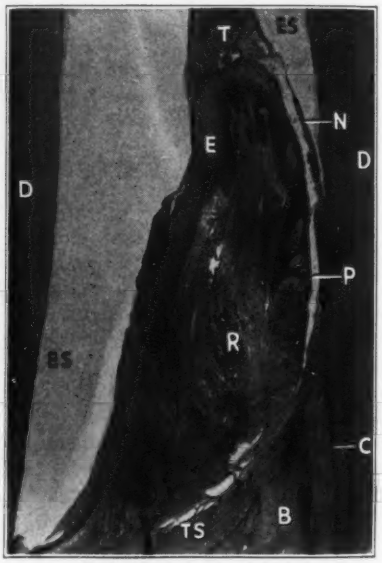

Fig. 2.—Gingivitis. D, dentin; T, débris; E, epithelium; R, round-cell infiltration; ES, enamel space; TS, transeptal fibers; B, bone; N, Nasmyth's membrane; P, pocket; C, cementum. This section shows very early pocket formation.

CLASSIFICATIONS

The microscopic study of necropsy material collected and sectioned disclosed four types of parodontal disease. The classification is based on histopathology and is further substantiated by clinical investigation.

1. *Simple Gingivitis.*—There is no bone involvement. The condition is due to malhygiene, faulty fillings and systemic conditions, such as pregnancy and dysmenorrhea.

2. *Marginal Parodontitis.*—This is a

localized condition such as has been described by Gottlieb as schmutz-pyorrhea, and by others by different names. In this type, there are marked bony changes affecting the alveolar crest.

3. *Parodontosis (Generalized Involvement).*—This is of systemic origin, starting in the bone. It is a reversion of bone into connective tissue.

4. *Syndrome of Marginal Parodontitis and Parodontosis.*—This is a combination of the preceding two, which may occur in various combinations.

Fig. 3.—Marginal parodontitis, pocket formation. D, dentin; C, cementum; T, calculus and débris; P, pocket; TS, transeptal fibers; B, bone; M, marrow; PM, peridental membrane; E, epithelium; R, round-cell infiltration. This section shows pocket formation with calculus in the pocket. Also shown are the transeptal fibers which are broken and new ones forming underneath.

HISTOPATHOLOGY

1. *Simple Gingivitis.*—As the name implies, gingivitis is an inflammation of the gingiva. The usual clinical symptoms are redness and tendency to hemorrhage. There are no pockets formed, nor is pus present. Microscopic study shows that the epithelium is attached to the tooth at the cemento-enamel junction. The epithelium usually shows slight papillary downgrowth into the gingival corium. A deposit attached to the tooth in the gingival crevice may be found, as shown in Figure 1. Round-cell infiltration in the connective tissue is a feature. This infiltration extends down to the transverse fibers, which remain intact. The capillaries are enlarged and hyperemic. Usually, there

is no pocket formation, but if this does occur, it is incipient, as in the left pocket seen in Figure 2. If the process is of long standing, there is a proliferation of connective tissue which leads to a hyperplastic condition.

2. *Marginal Parodontitis.*—This is the local type of parodontal disease in which the symptoms are in keeping with the classic descriptions of pyorrhea alveolaris.

Fig. 4.—Pocket formation, downward growth of epithelium. D, dentin; C, cementum; P, pocket; E, epithelium; R, round-cell infiltration. This section shows epithelial proliferation and pocket formation. It shows also how part of the gingival corium may be desquamated.

This type of disease starts at the alveolar crest and works down until the alveolar walls are resorbed and the tooth is exfoliated. Thus it can be seen that the first changes occur in the gingivae and that the bone becomes involved later.

Epithelial changes. The changes in the epithelium consist of proliferation and a downward growth. It extends into the underlying gingival corium in finger-like projections, which frequently anastomose. As they anastomose, small portions of the corium are surrounded and these portions atrophy, bringing the epithelium, which previously grew down, to the surface. The epithelium also tends to grow down along the cementum, and thus the pocket, increasing in depth, becomes continuously lined with it. The epithelial proliferation also is ahead of the destructive process, and therefore ulcers rarely form, though the proliferated surface epithelium may be desquamated. A mass of débris made up of cellular structures and food (vege-

table matter and muscle fibers) is seen lying in the pockets. Usually, calculus is attached to the tooth. (Fig. 3.)

Changes in the submucosa. In the gingival corium, the most significant change is the round-cell infiltration. The cells are very numerous and sometimes densely packed together. Both lymphocytes and plasma cells are seen. In some sections, the connective tissue of the corium is entirely obliterated by this round-cell infiltration; whereas, in other specimens, there is much less cellular response. In these specimens, leukocytic infiltration can be seen around the blood vessels only. An observation has been made that the greater the amount of injury to the bone, the greater the cellular infiltration.

The transverse fibers which pass just above the crest of the bones are seen in

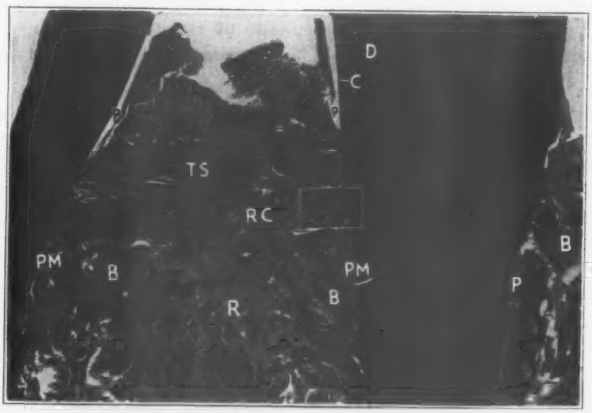

Fig. 5.—Marginal parodontitis. P, pocket; D, dentin; C, cementum; TS, transeptal fibers; PM, peridental membrane; B, bone; R, resorption; RC, round-cell infiltration. This section shows resorption of the alveolar process starting at the crest and working down.

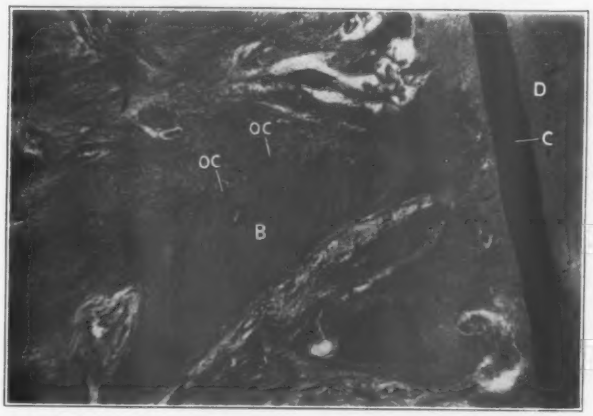

Fig. 6.—Marginal parodontitis. (High power section of Figure 5.) D, dentin; C, cementum; B, bone; OC, osteoclasts. This shows lacunar resorption with the presence of osteoclasts, as found in marginal parodontitis.

all stages of the disease. When the fibers are destroyed, new ones grow underneath and take the place of the original ones. (Fig. 3.) Frequently, the cellular infiltration extends into these transverse fibers and even into the marrow spaces of the bone beneath.

Changes in the peridental membrane. The peridental membrane has, in the past, been considered very important in parodontal diseases. This attitude was based on clinical observations and was well founded. However, in the marginal type of parodontal disease under discussion, the peridental membrane is not affected except by a gradual progressive disintegration as pocket formation increases and bone is resorbed. Otherwise, there was no evidence of alteration in our histologic sections of this type.

Pocket formation. In marginal parodontitis, the pockets are shallow, since the bone is lost at the crest and not along the side of the tooth. As the alveolar crest fibers are always maintained, vertical pocket formation is prevented. Vertical pockets may be found in the marginal type because of (1) a combination of systemic and marginal types of disease, which will be explained later and (2) the effect of malocclusion or some other local condition. Malocclusion causes an ab-

Fig. 7.—Parodontosis. D, dentin; C, cementum; P, pocket; B, bone; PM, peridental membrane. This section shows vertical pocket formation and also reversion along the membrane of the left tooth. The epithelium has proliferated down to the lower border of the large rectangle. This will keep lining the pocket to its depth as it progresses.

normal occluding contact of the teeth, which, in turn, causes resorption of bone on one side of the tooth, resulting in vertical pocket formation. Also, mechanical irritation, such as that from an overhanging filling or from food impaction, may destroy the alveolar crest fibers, allowing infection to attack the bone.

Changes in the bone. The bone changes, which are secondary, are caused by the infection and irritation that take place in the gingival corium. The process is that of lacunar resorption and the presence of osteoclasts in Howship's lacunae may be frequently observed, although osteoclasts may be absent when an indented surface only is presented. The crest of the alveolar bone is opened by this process of resorption and generally has a pitted appearance. The deep portion of the socket is affected only in the very advanced stages of the disease.

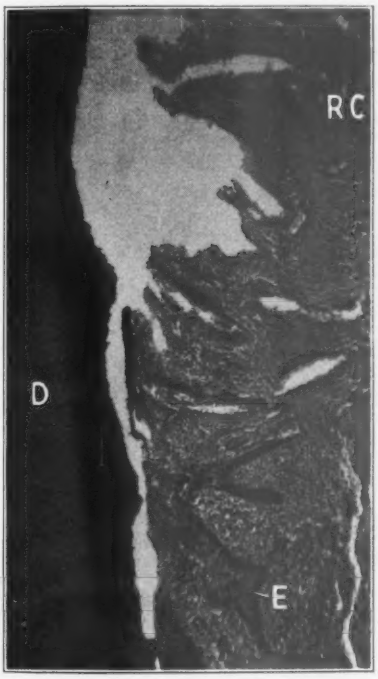

Fig. 8.—Parodontosis. D, dentin; E, epithelium; RC, round-cell infiltration. This section shows how the epithelium has proliferated along the pocket. There is resorption of the cementum. (High power magnification of Figure 7.)

3. *Parodontosis.*—Parodontosis is the most important of parodontal diseases. Clinically, it is recognized by the loosening and wandering of the teeth. It is of systemic origin and the bone is affected first. Parodontosis is therefore a disease of the alveolar bone in which the bone is transformed into fibrous tissue by reversion. In the early stages, the gingiva may or may not be affected by pocket formation. This usually occurs later, however.

Bone changes. The main pathologic change in parodontosis was found to be bone reversion. This is a process which seems to cause the disappearance of bone without apparent intervention of cellular elements, such as osteoclasts. According to Leriche and Policard, "Osteolysis or bone reversion is a true disintegration of bone, which reverts to connective tissue." They think that there is a rupture of the chemical balance in a direction opposite to the fundamental ossifying disposition. One factor active in bone reversion seems to be pretty generally accepted; namely, an increased circulation of the blood. Formerly, it was thought that bone atrophy was caused by insufficient circulation, but more recent studies seem to disprove this theory. Our own studies confirm the fact that the circulation is increased in bone reversion, blood vessels being found in increased number and greatly enlarged. In bone reversion, the inorganic substance of the bone is lost and the bone cells are liberated, but the fundamental fibrous connective tissue network remains. This tissue may be increased in density by fibroblastic activity of the liberated osteoblasts that have undergone reversion. Thus, the bone ceases to be bone, but the matrix persists as connective tissue; whereas, in osteoclastic resorption, the bone is replaced by the ingrowing neighboring granulation tissue.

The initial lesion in parodontosis is in the alveolar bone around the teeth. Here, we see the bone reversion. The bone disintegrates, leaving osseous islands surrounded by dense connective tissue. In other instances, resorption takes place in the center of trabeculae in which large blood vessels can be seen. (Fig. 2.)

The marrow of the alveolar bone presents a very interesting picture in this disease. The normal fatty marrow becomes fibrosed. We see loose connective tissue with numerous dilated blood vessels. The bone marrow of the jaw itself, however, shows the normal picture.

a protective reaction against loosening of the tooth, which may also be the cause of the formation of cementicles in the peridental membrane, as well as tears in the cementum, both of common occurrence in this type of parodontal disease.

The peridental membrane. This structure is always free from inflammation. If there is pocket formation and inflammation, cellular infiltration is limited to the parts nearest the inflammatory area. There is no further progress of inflammatory infiltration without the previous resorption of the bone. A characteristic change, however, which affects the entire

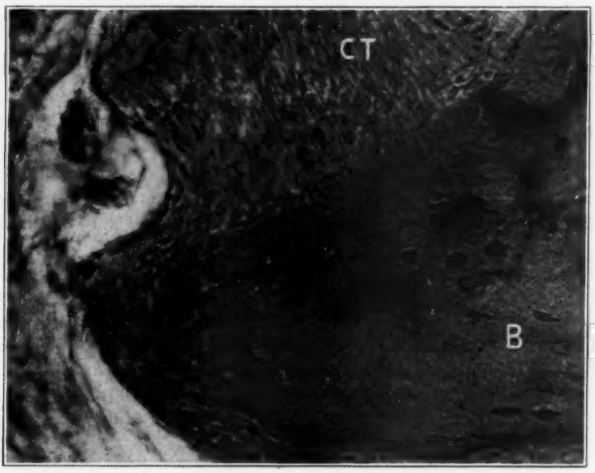

Fig. 9.—Parodontosis. B, bone; CT, connective tissue. This section is a high power photomicrograph showing how the bone cells are liberated and how bone reverts to fibrous tissue. (High power magnification of Figure 7.)

The cementum. In parodontosis, the cementum of the teeth shows interesting changes. Hypercementosis is usually produced, the amount apparently being in direct ratio to the amount of bone resorbed. As a rule, the greater the amount of bone lost, the greater the amount of secondary cementum formed, and the tighter the tooth. If there is little production of cementum with a great loss of bone, the teeth are very loose. Therefore, we have interpreted this phenomenon as

peridental membrane is the dilation of the blood vessels and also a proliferation of the capillaries. Often, the capillaries are seen to be growing at right angles to the bone and entering it, an evidence of vascular bone resorption.

Motility. The alveolar wall adjacent to the membrane is resorbed, as described above, and, as a result, the peridental membrane is widened and the tooth becomes loose. The peridental membrane is affected more at the apical and crest

portions than at the middle. The resorption at the apical portion results in a thick membrane, which gives the tooth much leeway in motion and allows it to move as in a ball-and-socket joint. Thus, the wider the peridental membrane becomes, the more chance there is for movement of the tooth, and the more movement, the looser the tooth becomes, and so cause becomes effect, and effect, cause.

Pocket formation. It has long been observed by many investigators that parodontal disease may progress in two ways. Thus, they differentiate the disease into two types, one with horizontal and one with vertical pocket formation. This

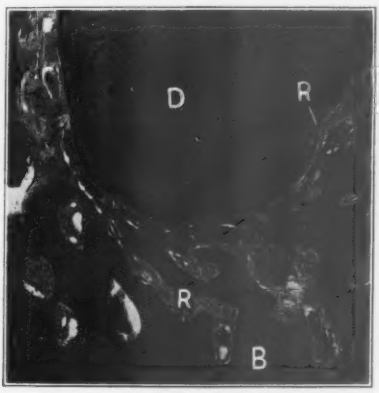

Fig. 10.—Parodontosis. D, dentin; R, resorption; C, cementum; B, bone. This section shows resorption of the tooth and bone, and the process of widening of the peridental membrane. (Compare with Figs. 7 and 12.)

differentiation was based on clinical observation. In parodontosis, the reversion of the alveolar bone takes place to the most marked degree next to the peridental membrane, and it is this widening of the membrane which allows the vertical pocket formation. The alveolar crest fibers entering into the bone are lost; which causes the gingival margin to relax, and allows pocket formation. Since the attachment of the principal fibers to the bone is weakened, owing to the widening of the peridental membrane, pocket formation progresses in a downward direction. Therefore, in parodontosis, the pockets are deeper than in the local or marginal parodontitis.

Occlusal function. The alveolar bone is a structure easily affected by functional conditions. It has been known for a long time that changes in occlusal function will produce changes in the architecture and structure of the alveolar bone. In the jaws, therefore, we have a functional factor not found in other bones, which may

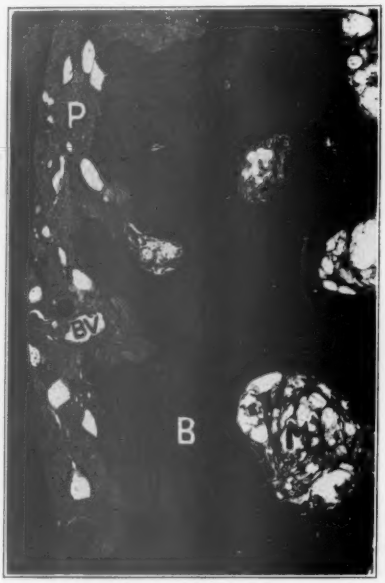

Fig. 11.—Parodontosis. P, peridental membrane; BV, blood vessel; B, bone; M, marrow. There is marked increase in the number and size of blood vessels in the peridental membrane.

explain why, in parodontosis, other bones are not involved. Also, the alveolar process, on account of the peculiarity of its blood supply, is an end organ, as is the marginal gingiva, and its structure is very labile. It is the only bone that may change to such a marked degree, because it forms in response to dental function

and resorbs when function ceases, changing all the time in response to occlusal requirements.

In our studies, we have come to the conclusion that, in parodontosis, the bone substance cannot be maintained in normal condition owing to the effect of poor nutrition or toxic processes on the bone cells. Instead of responding normally to function by strengthening, the bone undergoes disintegration. In some cases of normal function, the bone may just be able to maintain itself; but if teeth become loose, functional force is increased and becomes too great a strain. Thus, a vicious cycle is established, the increase in functional stress producing resorption, and resorption, weakening the attachment of the tooth, increasing functional stress. In this way, altered occlusal function can be the cause of more rapid alterations in the bone.

4. *Syndrome of Parodontosis and Marginal Parodontitis.*—In studying our material, we find that the combination of parodontosis and marginal parodontitis is very prevalent. The common occurrence of this syndrome probably accounts for the fact that many cases clinically diagnosed as marginal parodontitis do not react satisfactorily to treatment. The combination may also account for the fact that we see vertical pocket formation in cases supposedly of the marginal parodontitis type. The syndrome may develop (1) in a long standing case of marginal parodontitis wherein parodontosis sets in as a separate phase; (2) in cases wherein marginal parodontitis and parodontosis begin to develop at the same time, and (3) in cases of advanced parodontosis in which the marginal parodontal structures succumb.

Pathologic development. The two main processes described are found in this syndrome. All the features of parodontosis can be seen and, at the same time, the changes of marginal parodontitis.

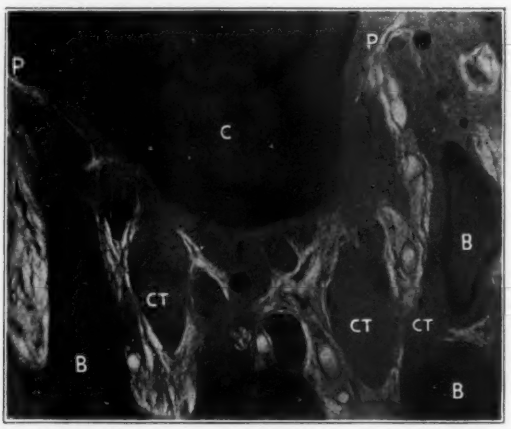

Fig. 12.—Parodontosis. P, peridental membrane; CT, connective tissue; B, bone; C, cementum. This section shows marked widening of peridental membrane. Bone reversion is evident and former whole spicules of bone are now connective tissue.

1. In advanced marginal parodontitis plus early parodontosis, the pathogenesis can be explained as follows: The patient has been afflicted by the local marginal disease alone, until his systemic condition changes, when the pathologic changes of parodontosis set in. Such cases are almost impossible to diagnose clinically, since the subjective and objective symptoms of marginal parodontitis entirely

overshadow the systemic parodontal condition.

In microscopic study, we see the pathologic process of marginal parodontitis, previously described. Along the wall of the alveolus, however, the following bone changes can be observed: bone reversion into fibrous tissue, a slight fibrosis of the marrow and hyperemia of the blood vessels. The peridental membrane has also

other parodontal diseases. The clinical diagnosis will usually be that of a simple gingivitis because gingivitis is the beginning of marginal parodontitis. In the early stages of parodontosis, no subjective or objective symptoms occur that we may recognize as yet; therefore, this part of the syndrome is overlooked.

Study of the histopathology of this condition disclosed many interesting con-

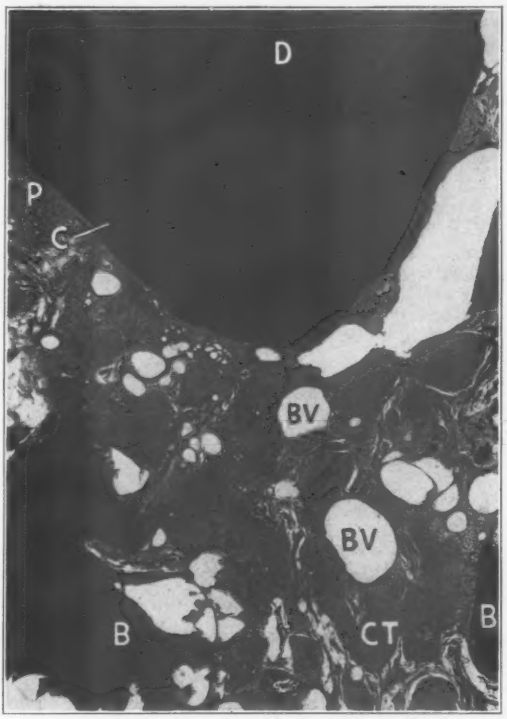

Fig. 13.—Parodontosis. D, dentin; C, cementum; P, peridental membrane; B, bone; BV, blood vessel; CT, connective tissue. Increase in number and size of blood vessels and widening of peridental membrane are evident. The process of gradual reversion is well shown around the two bone trabeculae marked B on the right.

numerous blood vessels which are engorged. The membrane itself is widened only slightly in this type of case.

2. In early marginal parodontitis and early parodontosis starting simultaneously, we have a combination which is perhaps more often wrongly diagnosed than all

ditions. At the apical portion of the alveolus, reversion of the bone is evident, resulting in a widening of the peridental membrane. Changes at the crest show both lacunar resorption and bone reversion. At the crest, the external border is usually attacked by osteoclastic resorption

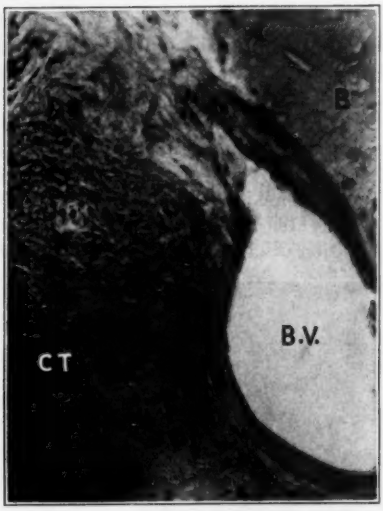

Fig. 14.—Parodontosis. B, bone; CT, connective tissue; BV, blood vessel. This section shows in high power magnification the characteristic connective tissue that is formed. Large blood vessels are seen in the area.

proliferation, lining the pocket and, from its base, growing down alongside the cementum. There is marked round-cell infiltration in the gingival submucosa.

3. In advanced parodontosis, pockets form and local etiologic factors enter the picture. It must be borne in mind that, as soon as there is pocket formation, infection and irritation set in, and the two processes are probably evidence of late stages of parodontosis. This condition may be recognized clinically by the loosening and wandering of the teeth. The gingival tissues are usually swollen and have a reddish discoloration. There may be pus discharge from the gingival margin at the orifice of the pockets. In some cases in which drifting of the teeth does not occur, it is difficult to distinguish this condition from marginal parodontitis.

SUMMARY

The object of this report is to record new evidences of the histopathologic process in parodontal diseases based on a study of sixty-seven jaws obtained at necropsy. The classification comprises four types: (1) simple gingivitis, (2) marginal parodontitis, (3) parodontosis and (4) the syndrome of marginal parodontitis and parodontosis. In this paper,

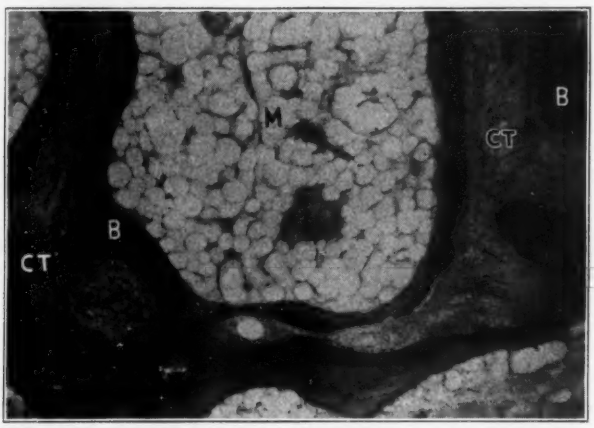

Fig. 15.—Parodontosis. B, bone; CT, connective tissue; M, marrow. This section shows bone reversion inside the bone trabeculae transforming them into fibrous tissue.

alongside the peridental membrane. Osteoclastic resorption is not very striking in these cases. It was seen in only a few instances and nowhere except at the alveolar crest. Pocket formation is evident and the epithelium shows a downward

we have endeavored to describe the pathologic processes of each type. We hope in future papers to correlate the pathology of parodontal diseases with the clinical symptoms, and also to relate the medical histories of the patients whose jaws were obtained to oral disease.

Fig. 16.—Syndrome of parodontitis and parodontosis. P, pocket formation; CL, calculus; B, bone; M, marrow. The section shows that there is calculus in the pockets, causing marginal parodontitis, while parodontosis affects the alveolar bone.

Fig. 17.—Syndrome of parodontitis and parodontosis. D, dentin; C, cementum; CT, connective tissue; R, reversion; B, bone; OS, osteoclasts. Higher magnification of the section shows the two types of bone resorption present. Bone resorption with the presence of osteoclasts is evident, as well as bone reversion into fibrous tissue. This section is at the alveolar crest.

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OUR CHANGING CONCEPT REGARDING PULPLESS TEETH

By LOUIS I. GROSSMAN, D.D.S., Dr. med. dent. (Rostock), Philadelphia, Pa.

ALMOST three decades have passed since the medical and dental professions were startled into action by Hunter's denunciation of oral sepsis. It is time now to take stock of what has been said and what has been done since that time in order that we may know where we stand today and not repeat the errors of the past.

MISCONCEPTIONS REGARDING PULPLESS TEETH

Since 1910 two popular misconceptions have persisted in the minds of dentists and physicians alike, namely: (1) that by oral sepsis Hunter meant abscessed teeth; (2) that a pulpless tooth was a "dead" tooth. I should like to set aside these misconceptions at once, so that we may understand each other the better.

In an editorial in THE JOURNAL OF THE AMERICAN DENTAL ASSOCIATION[1] entitled "A Persistent Misconception About Hunter and the Subject of Focal Infection," the Editor refers to Hunter's pronouncement as follows: "The 'oral sepsis' of which he complains—and not without reason—had nothing whatever to do with the thing that, during the decade that followed his address, caused the greatest concern among the profession, i.e., focal infection from the apical ends of pulpless teeth. This is the thing that claimed our major consideration, and in connection therewith the name of Hunter has repeatedly been quoted. As a matter of fact, Hunter never referred in the remotest way to the evils of pulpless teeth as such. He was concerned with the sepsis that came from accumulations around crowns, bridges, and artificial dentures, calling them 'gold traps of sepsis.' . . . Mayhap if this distinguished scientist had given his whole-hearted attention to this important question, the mental aberration and almost universal prejudice against the pulpless tooth that has developed might have thereby to a certain degree been avoided." That this misconception could have been perpetuated for so many years is indicative of the hysterical era which followed Hunter's criticism. Had his words been taken to mean what he intended them to, the extraction orgy of the past might have been averted.

The second misconception, that a pulpless tooth is a dead tooth, can be quickly

(Read before the Section on Histology, Physiology, Pathology, Bacteriology and Chemistry [Research] at the Seventy-Ninth Annual Session of the American Dental Association, Atlantic City, N. J., July 14, 1937.)

dispatched by the application of the pragmatic principle. One need only place the beaks of forceps on a pulpless tooth in an effort to remove it, to realize that what is sometimes considered "dead" is still very much alive. A pulpless tooth is *not* a dead tooth! It still has a nutritional supply. It still has a vital relationship with the surrounding tissue. The living body does not tolerate dead tissue; it exfoliates it. Pulpless teeth are not exfoliated; they are not dead.

I should like to call your attention to the fact that there are fashions in the healing, as well as in the sartorial, art. A little more than 50 years ago hysterectomy was fashionable, and about thirty-five years ago nephropexy was in style. Many systemic ills, from simple fatigue to gastric disturbances and cardiac palpitation, were attributed to the movable kidney. Commenting on focal infection in the light of the past, Dr. O. H. Perry Pepper[2] said a decade ago, "It is instructive and humiliating to go back into the literature of twenty-five years ago and read the many articles describing the variety of conditions and symptoms attributed to the movable kidney and the remarkable therapeutic results obtained by nephropexy." He then asks the question, with regard to focal infection, "Are we merely in the midst of a movement which will run its course and be looked back upon in ten years as we look back on the days of nephropexy?"

DENTAL FOCAL INFECTION

We have happily passed the peak of the focal infection wave. That many have lost their teeth without systemic improvement, none will deny. But it is equally undeniable that the focal infection scare had a very desirable effect in that it focused the attention of the two professions on the potential possibilities of grossly infected teeth causing systemic harm and in furthering research work on the relationship of pulpless teeth to systemic disturbances. Among such outstanding work is that of Rosenow,[3] who frequently succeeded in showing a focal infection relationship by animal experimentation. His work, however, has not been generally accepted: (1) Because of the massive doses of inoculant injected, as much as 10 cc. being used at one time. An equivalent amount, if man were the laboratory animal, would be the injection of about one pint of inoculant. (2) Because the intravenous route was generally used for the injections. (3) Because the organisms were at the height of virulence at the time of injection. Furthermore, it has been asserted that Rosenow's work has not been adequately controlled, and that the technic employed by him so devastates the laboratory animal that lesions are sometimes produced in almost every tissue and organ of the body. Rosenow's conclusions have not been entirely confirmed, and are looked upon with much skepticism if not with actual disfavor by some bacteriologists.

It is perhaps significant that the popularity of the focal infection theory was greatest in this country, where it was nurtured and publicized, and that its importance has been minimized or practically disregarded in Europe.

It is also significant perhaps that no definitely conclusive proof has yet been advanced that in any single case a tooth or teeth were the direct cause of the systemic disturbance, excepting *a priori* proof. The extraction of a tooth followed by amelioration of symptoms does not necessarily prove that the tooth was the cause of the disease. This is only an assumption which may or may not be true. In the years between 1910 and today, many teeth were sacrificed without improvement in the general health of the patient. Even Charles H. Mayo[4] sounded a note of caution, as far back as 1928, regarding indiscriminate removal of teeth.

At that time he said, "A few years ago there was a period of attributing rheumatism and many other diseases of nerves,

joints, muscles and the heart to diseases of the teeth which might be eradicated. This is all very true, *but* without careful investigation, in many instances, good teeth were sacrificed without improvement in health. . . . The good dentist today has learned that there are other foci and advises further examination before the extraction of apparently healthy teeth."

In the most comprehensive review of the literature on focal infection published up to this time, MacNevin and Vaughn[5] analyze the evidence for and against focal infection and conclude that the proof submitted in favor of the theory is not altogether convincing. They also point out that the evidence of animal experimentation submitted by Price, Rosenow and others, has its limitations from the standpoint of both technic and interpretation of results. The interpretation of such experimental work, they say, "must be pursued with extreme caution and openmindedness. The deductions often made are unwarranted."

THE PULPLESS TOOTH

Let us briefly review the more important evidence relating to *periapical* focal infection which has accumulated in the last twenty-five years. Let us examine it critically. If necessary, let us be prepared to change our attitude toward pulpless teeth. The accumulated evidence may be divided arbitrarily into four categories: (1) roentgenologic; (2) bacteriologic; (3) histologic; (4) clinical.

We shall first consider the roentgenologic studies made of pulpless teeth.

Roentgenologic Studies.

(1) Both roentgenologic and clinical records of nearly 2,000 patients have been carefully studied at the University of Illinois.[6] The status of these pulpless teeth has been followed up over a period from 1 to 16 years after completion of treatment. At the last tabulation in 1936, 76 per cent either had remained or were *roentgenologically negative* throughout the period of observation. In this group were included teeth with acute and chronic abscesses, and with granulomas.

(2) Stein,[7] of Vienna, made a roentgenologic study of 223 pulpless teeth over a period of from 2 to 8 years after treatment. Those with areas of rarefaction improved in 44 per cent of cases, while those without areas of rarefaction remained normal in 76 per cent of cases. We shall now consider the bacteriologic studies.

Bacteriologic Studies.

A number of important bacteriologic studies of pulpless teeth have been made. The findings, unfortunately, are not in close agreement. The technic of taking samples for culturing, the culture media used, the method of culturing and examining for bacterial growth and the number of teeth studied undoubtedly account for the discrepancies. While Appleton is of the opinion that the most desirable method of taking a sample for culturing is by the "external approach" method, only one investigator, Burket,[8] has used this method in a large number of cases. The other method of culturing most commonly used is that following the extraction of a tooth.

Haden,[9] together with others, has used the latter method extensively, reporting on a series of 1,100 pulpless teeth. Inasmuch as Haden's work is representative of one method of approach, and Burket's work is representative of the other method, we shall examine the findings of both. Haden obtained growth from 87 per cent of pulpless teeth, while Burket obtained growth in only 72 per cent. Haden also obtained growth from 55 per cent of vital teeth, and Burket obtained growth from 43 per cent of vital teeth. Although there is a difference of from 12 to 15 per cent, the findings run fairly close together considering the fact that the methods differed. Let me now make this very important observation: *If the vital teeth are used as controls, the difference between the percentage of growth*

obtained in the pulpless and in the vital teeth will then give us the actual percentage of growth found in pulpless teeth. Averaging this percentage from the data of Haden and Burket, we find that growth was obtained in only 30 per cent of pulpless teeth, and *growth does not necessarily mean infection.*

When we take into consideration the fact that the teeth examined were from people "taken off the street" so-to-speak, i.e., neither the quality of the work done nor the number of years since it was done are known, this relatively low percentage of growth from pulpless teeth seems all the more remarkable. Obviously, root-canal work done thirty or even twenty years ago would probably not be so good as that done today. Furthermore, it is doubtful if many, or even any, of the teeth treated had the benefit of a smear or culture to determine sterility of the root canal before it was filled. That these are important and pertinent factors which bear strongly on the fate of the bacteriologic outcome none will deny. The data of Rhein, Krasnow and Gies, as interpreted by Appleton,[10] and the studies of Grossman,[11] suggest strongly that a bacteriologic examination before filling the root canal assures the tooth a better chance for success as determined afterward by roentgenologic and clinical, or bacteriologic studies. It is conceded that the pulpless teeth examined by Haden, Burket and others represented a cross section of the pulpless teeth in the mouths of most people at the time the studies were made. What I am trying to say is that since the number of dentists using bacteriologic control is increasing, the bacteriologic picture of the pulpless tooth of the present and of the future is more encouraging than that of the past. This point we shall consider again later on.

There is one more thing that should be emphasized here in a discussion of bacteriologic data. The finding of bacterial growth from a sample taken from apical or periapical tissue does not necessarily imply that infection is present. Microorganisms are not pathogenic by themselves. They are pathogenic only in relation to the host. The mere presence of bacteria does not constitute infection. Hiss and Zinsser[12] say, "Bacteria are ever present in the environment of animals and human beings and some find lodgment on various parts of the body. The mouth, the nasal passages, the skin, the upper respiratory tract, the conjunctivae, the ducts of the genital system, and the intestines are invariably inhabited by numerous species of bacteria." They go on to say further, "It is evident, therefore, that the production of an infection must depend upon other influences than the mere presence of microorganisms and their contact with the body." They sum up the nature of infection by saying, "The inciting or inhibiting factors which permit or prohibit an infection are dependent in part upon the nature of the invading germ and in part upon the conditions of the defensive mechanism of the subject attached. Even after bacteria of a pathogenic species, in large numbers and of adequate virulence, have passed through a locally undefended area in the skin or mucosa of an animal or a human being by a path most favorably adapted to them, it is by no means certain that an infection will take place."

What then is the significance of the bacteriologic findings of pulpless teeth? If we make too literal an interpretation we may have to extract all teeth, both vital and pulpless. For example, Haden is of the opinion that chronic infection occurs quite commonly in *vital pulps* and adds "many believe there is a chronic infection in the pulp of every tooth in which the dentin is invaded by caries." If we took this literally we should have a toothless nation, because it is estimated that more than 90 per cent of the people of the nation have carious or have had carious teeth at one time. But is this literal interpretation of the presence of

microorganisms justified? It would seem almost as if some of the dental critics had a peculiar strain of bacteriophobia. Certainly they make no such literal interpretation regarding the presence of the pneumococcus in the mouth, and yet the *Diplococcus pneumoniae* is very commonly found in the mouth.

Who knows but that in the limited state of our knowledge today, the root-canal problem is not entirely a bacteriologic one, but an immunologic one? At the present time it would seem that bacteriologic studies of pulpless teeth are valuable in themselves only as they are correlated with clinical, histologic and roentgenologic studies.

Histologic Studies.

Histologic evidence of periapical repair following root-canal therapy has been furnished by Blayney,[13] Coolidge,[14] Kronfeld,[15] Aisenberg,[16] Hatton, Skillen and Moen,[17] Orban,[18] Gottlieb, Schwarz and Stein.[19] Skillen,[20] for example, studied histologic sections of 250 pulpless x-ray negative teeth. In only 6 teeth of this number did he find any evidence of infection. Instead of showing lantern slides depicting periapical repair, we might better utilize the time in considering what goes on in the periapical region during the reparative period.

Hatton[21] interprets the changes following successful root-canal therapy in the light of repair following bone injury or fracture: (1) A destructive change; (2) a constructive change; (3) a resting stage. The first stage (1) is characterized by "removal, resorption and dissolution of all the tissue damaged to the extent that it cannot function, whether it be in the pulp canal or in the apical space." The next stage (2) is characterized by "replacement of lost hard tissues of the tooth by osteoid cementum, and bone by new bone." This calcifying process occurs in the accessory canals as well as in the main root canal and extends out into the periapical tissues. (3) When the calcifying process ceases, the resting stage is reached. In this, the condition of the periapical tissue "persists for years without evidence of any change, inflammatory or otherwise, except for a tendency of these soft tissues to undergo a fibrous atrophy. . . . Since the reactions at the apex of a pulpless tooth are essentially similar to those found in the healing of a broken bone, it is assumed that they are physiologic in character."

Clinical Studies.

Lastly, we shall consider the clinical evidence bearing on the root-canal problem.

(1) It is a well known fact that a very large number of people having pulpless teeth show no apparent systemic involvement.

(2) Frankel[22] studied the incidence of systemic disturbances in a large group of people with and without so-called "heavy dentistry." By this term he meant the presence of crowns, bridges, pulpless teeth and any other evidence which might be suspected of indicating a focus of infection. He concluded that the incidence of systemic disturbances in the "heavy dentistry" group was not materially higher than in the other group.

(3) During the World War draft, data were collected of more than two and one-half million men between the ages of 18 and 30 years. An evaluation of the data, according to Appleton,[23] shows that "chronic infection about the head is not . . . the sole determining factor in the contemporaneous occurrence of such conditions as gastric ulcer, appendicitis, endocarditis, arthritis, osteitis deformans, myositis, and muscular rheumatism."

(4) A different kind of clinical evidence bearing on the status of the pulpless tooth is presented by Bryant and Polevitzky.[24] Blood picture studies were made in two groups of people with pulpless teeth: (1) In Group I, root-canal therapy was carried out in the usual manner; (2) in Group II, instead of root-canal therapy, the teeth were extracted. Modified Arneth blood counts were made at stra-

tegic periods of time before and after root-canal therapy or extraction, in order to determine whether the focus of infection was eliminated. Bryant and Polevitsky came to the conclusion that "in the cases studied there has been more improvement shown in the chemotherapeutic than in the surgical cases."

Now that the case for root-canal therapy has been presented in a rather favorable light, I do not wish to give the impression that *all* pulpless teeth should be treated and saved. I feel, however, that it was necessary to present the evidence in this way in order to down the doubting Thomas who, perhaps because of personal shortcomings, questions whether *any* root-canal work should be done. As may be expected, the rational stand is somewhere between these two extremes. What of the future of pulpless teeth? It is my strong belief that the future of pulpless teeth depends not so much on whether one antiseptic agent or another is used, but (1) upon the judicious selection of cases and (2) upon a bacteriologic examination of the tooth before filling the root canal in order to determine whether sterility has been attained.

SELECTION OF ROOT-CANAL CASES

In my own practice I have tried to follow six simple rules for the selection of cases for root-canal therapy. These rules are in the nature of "Don't's," or contraindications for root-canal therapy. They are as follows:

No root-canal therapy is done in the following:

A. General Systemic Factors.

1. In patients having arthritis, rheumatic fever, iritis, cardiovascular-renal diseases (the focal infection group of diseases).

2. In patients having leukemia, syphilis, active tuberculosis or diabetes.

B. Local Factors.

1. Where extensive apical involvement is present, either of periapical tissue or the cementum of the tooth.

2. Where extensive paradental involvement is present, e.g., alveolar resorption affecting one-half or more of root surface.

3. Where periapical region is inaccessible through apical foramen either because of mechanical obstruction, shape, or size of root canal.

4. Where an aseptic technic cannot be carried out.

It is doubtful if dentists of 20 or 30 years ago selected their root canal cases. Had they done so, it is entirely possible that pulpless teeth would never have been subjected to so much criticism. Whether pulpless teeth will be criticized in the future will depend not only upon the selection of cases, but also upon a bacteriologic examination to determine if the root canal is sterile, before filling it. I have purposely left the discussion of bacteriologic control of root canals for the last because of its importance and because I wish to give it special emphasis.

BACTERIOLOGIC EXAMINATION OF PULPLESS TEETH

If the object of root-canal therapy is sterilization, what folly it is to look at the dressing, smell it, and so decide whether the canal is ready to be filled. And yet, I daresay 90 per cent of those practising root-canal therapy do that very thing year in and year out. That there are not more failures is the surprising thing. In a study I made with the cooperation of a number of dentists, I[11] found that bacteria were still present in 42 per cent of cases when no bacteriologic criteria were used before filling the root canal. The usefulness of bacteriologic examination in root-canal work is also stressed by Appleton.[10] His conclusion, based on statistical data, is that "A case of periapical infection which yields a negative culture before filling the canals has a more favorable chance of clinical and roentgenographic success than does a case whose canals are filled while the cul-

tures are still positive." With only slightly better than an even chance to guess right, why guess? Why subject the patient to the risk, when a bacteriologic examination, either by smear or culture, is so simple and inexpensive a procedure?

It is not within the scope of this paper to discuss the technic of a bacteriologic examination. The procedure is far less complicated than taking and developing an x-ray picture. If necessary, it could be done by an intelligent dental nurse. It is not lack of knowledge which is blocking the way to intelligent and successful root-canal work, but lack of realization that such knowledge is at hand and can readily be applied. It is no longer a stigma to save pulpless teeth. It is a stigma to practise root-canal therapy indiscriminately and without due regard for asepsis. And it is a stigma to remove teeth that might be retained with adequate root-canal therapy.

SUMMARY

I have endeavored to bring to the attention of the dental profession at this time (1) certain evidence that root-canal therapy may be considered a safe procedure, compatible with bodily health, if properly done and (2) the fact that the future success of root-canal operations will depend to a great extent on (a) the proper selection of cases and (b) upon a bacteriologic examination of the tooth to determine its sterility before filling the canal.

No dental problem links us more closely with the medical profession than the problem of pulpless teeth. It is my belief that the medical profession of the future will judge the progress of the dental profession to a large extent by the advances it has already made and will make in the field of root-canal therapy.

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Medical Arts Building.

THE RÔLE OF THE DENTIST IN THE CONTROL OF SYPHILIS

By R. A. VONDERLEHR,* M.D., Washington, D. C.

THE average dentist with the average practice, according to the laws of chance, will be called on to render dental attention to a syphilitic person in about one of every twenty patients who consult him.

The disease is of such a nature, however, that the intimate personal contact which takes place between the dentist and his patient is apt to result in the accidental transmission of the disease to the dentist.

Furthermore, the dentist as a professional man is naturally interested in the control of all of the communicable diseases, and syphilis is one of the most serious and most prevalent of them. Half a million people in the United States acquire syphilis and seek treatment annually. Another half million are infected, but fail to seek treatment. When such case rates are calculated on a cumulative basis, we learn that by the time the age of thirty-five years has been attained, from 5 to 7 per cent of the entire population of the country is or has been infected with this disease.

Syphilis is a disease characterized by the development of a primary sore at the point of inoculation; by the development, in most patients, of an eruption involving the skin and certain mucous membranes; by a more or less prolonged period of latency during which no symptoms are present and, finally, by the development of serious late crippling manifestations such as syphilitic disease of the heart and great blood vessels and involvement of the central nervous system. The period of the initial lesion, or primary syphilis, as it is commonly called by physicians, lasts for only a few weeks, at the beginning of the infection. This is followed in the average patient by an eruption which may last from a few weeks to a few months. If the patient's immunologic processes fail, owing either to natural conditions or inadequate treatment, the eruptive stage may reappear.

It is during the time when the chancre or the eruption is present that the dentist is in gravest danger in treating the infected person. Experience shows that the vast majority of acquired syphilis which is transmitted is spread during the first year of the disease, and that by the end of the fourth year, there is very little chance that the syphilitic patient will ever transmit acquired syphilis to any one except his progeny. The period of the initial sore and the eruption may, therefore, be said to define in a rough way the communicable stage of syphilis.

During the years of symptomless syphilis which follow, the patient is of little danger to those with whom he comes in contact. Female patients during latency offer a serious problem because of the frequency with which syphilis is transmitted to the unborn child. The complications of late syphilis, when the symptomless period ends, are of grave importance to the community because they so frequently offer serious problems in public welfare.

SYPHILIS AND THE ORAL CAVITY

One of the peculiarities of the infectious lesions of syphilis is that they have a special predilection for the oral cavity

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and the skin and mucous membranes adjacent to the mouth and nose. This fact frequently places the dentist in such a position that he may, if familiar with the lesions, be of material assistance to the physician in the detection of syphilis.

From the dentist's standpoint, syphilis may be considered under two general headings: 1. Early or communicable syphilis, during which the syphilitic patient is a potential danger not only to the dentist, but also to all others with whom he comes in contact. 2. Late syphilis, during which the disease in female patients may during pregnancy be transmitted to the offspring, in whom the possibility of transmitting acquired syphilis is negligible.

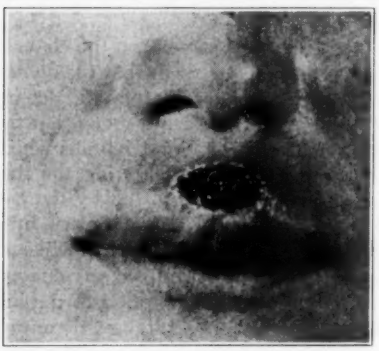

Fig. 1.—Chancre of cutaneous surface of upper lip.

COMMUNICABLE SYPHILIS

The two main infectious lesions which occur in or near the oral cavity are the chancre and the mucous patch. The latter type of lesion is in reality an eruption involving the oral mucous membrane.

The chancre, or primary sore, is the first recognizable lesion of syphilis. (Figs. 1-3.) It develops at the point where the germ of syphilis enters the body and is apt to occur in or near the oral cavity when the disease is transmitted by kissing or through perverted practices. The lesion itself is an eroded tumor which develops as a rule from ten to forty days after exposure. The diagnosis of syphilis cannot be made on the basis of the physical appearance of the sore since clinically indistinguishable lesions are frequently due to a number of other conditions. The most frequent location of the syphilitic chancre in the oral region is on the lips, tongue or tonsils. A common manifestation of primary syphilis is firm, rubbery, painless satellite enlargement of the lymphatic glands which drain the tissues on which the chancre is located. In the case of oral chancres, the glands at the angle of the jaw are those usually enlarged.

Fig. 2.—Chancre of mucocutaneous junction of lower lip.

In order to establish positively the diagnosis of primary syphilis, it is necessary to make a microscopic examination of the secretion from the sore. This examination, known to physicians as the dark-field examination, is one which requires special training and skill. If the first dark-field examination reveals the presence of the spirochete of syphilis, the diagnosis is certain; if the first examination is negative, it may be necessary to repeat it daily for several days. It is important that the chancre be not treated locally during the days of observation by dark-field examination. It is recommended that when the first microscopic examination is performed, a specimen of blood be taken in

order to perform a serologic test for syphilis. One of the greatest shortcomings of the serologic blood test, however, is the fact that during the first two or three weeks of the syphilitic infection, the reaction which indicates that syphilis is present does not occur. If the germ of syphilis cannot be detected after several dark-field examinations of the secretions from the sore, and if the first serologic blood test is negative, it is necessary to repeat these blood tests at intervals during the first three or fourth months in order absolutely to exclude syphilis as the cause of the ulcer. Fortunately, during this period when syphilis is present, the serologic blood test becomes positive in practically 100 per cent of the people who are infected.

Fig. 3.—Chancre of tongue. (Courtesy of J. E. Moore.)

It is a common occurrence some weeks after the appearance of the chancre for the syphilitic patient to develop a sore throat. This is among the first of the manifestations of syphilis of the oral cavity. It is commonly called syphilitic angina or syphilitic sore throat and is a diffuse inflammatory process which involves chiefly the pharynx and the tonsils. Its manifestations are not sufficiently clear-cut to permit even the experienced physician to recognize it beyond doubt without the use of laboratory methods. Fortunately, when this condition is present, the serologic blood test is positive in practically every patient.

THE MUCOUS PATCH

Few lesions indicative of so serious a disease are as innocent looking as is the mucous patch. Yet the mucous patch is one of the most typical lesions of the mucous membrane in early syphilis and is of vast importance because secretions from it fairly teem with the germs of syphilis. For this reason, the patient with mucous patches is in a very contagious stage. The typical mucous patch is a small circumscribed, slightly elevated and slightly inflamed lesion which averages from 5 to 10 mm. in diameter and is of a grayish or whitish color. (Fig. 4.) It may occur on the inner surfaces of the lips, the surface of the tongue and the buccal mucosa. Moisture and friction may cause the mucous patch to become eroded, and while the lesion presents a relatively typical picture to the experienced observer, the uninitiated person would tend to confuse a number of non-syphilitic lesions with it.

Fig. 4.—Hypertrophic mucous patches of lips. (Courtesy of J. E. Moore.)

The serologic blood test for syphilis has become such a commonplace procedure that even experts in syphilis now confirm the diagnosis of mucous patches by the use of such a test. As in other conditions

which occur in the eruptive stage of syphilis, the blood test is positive in practically every patient having a mucous patch.

ORAL LESIONS IN LATE SYPHILIS

The dentist, by developing a relatively high index of suspicion concerning syphilitic lesions in the mouth, can aid in the control of late syphilis by assisting to detect it before irreparable damage has been done and by bringing pregnant women to treatment in order to prevent the prenatal transmission of syphilis. There are three common late syphilitic lesions which occur in the oral cavity: (1) leukoplakia; (2) gumma of the oral cavity, and (3) Hutchinsonian teeth and other syphilitic dental defects.

Leukoplakia.—Syphilitic leukoplakia, from a pathologist's standpoint, consists of a condition which results from scarring following the healing of the mucous patch. It is a grayish, more or less sharply defined and somewhat thickened patch on a mucous or mucocutaneous surface. Irritation alone may produce a nonsyphilitic type of leukoplakia and predisposes to the syphilitic variety. The condition is relatively common in those who use tobacco or who have had bad teeth. One of the grave dangers, aside from the late crippling manifestations of syphilis, which sometimes follow in the wake of leukoplakia is the possibility that the condition may lead to malignancy. Patients in whom this condition is suspected should be referred to a physician for a thorough physical examination, including a serologic blood test and an examination of the spinal fluid.

Gummata.—These are the classical lesions of tertiary or late syphilis and most of the complications which occur in this stage of the disease are believed due to their development. Gummata may occur in any of the tissues of the oral cavity, but they are especially apt to appear in the soft and hard palates and in the tongue. If the patient is seen before the gumma ulcerates, the lesion is characterized chiefly by an area of swelling, which may be more or less inflamed. After a considerable period, the swelling tends to break down and may cause extensive destruction of tissue, ulceration and scar formation. *Fetor oris* and a nasal twang of the voice are common manifestations of gumma of the palate, the latter being present only when extensive perforation has occurred. These patients too should be referred to a physician for a complete physical examination and study of the blood and spinal fluid.

Dental Abnormalities.—Many dental abnormalities have been regarded as syphilitic, but findings in recent years indicate that dental abnormalities are frequently due to unknown defects of development, dental dystrophy and trauma. The most typical dental abnormality due to syphilis is the occurrence of the so-called Hutchinsonian teeth in patients with congenital syphilis. In Hutchinsonian teeth, the upper central incisors assume the shape of a truncated cone. The cutting edge of the tooth may be deformed by a crescentic notch, which is regarded as the most characteristic feature of the syphilitic incisor. The Hutchinsonian defect appears only in the second dentition.

Another dental abnormality frequently due to syphilis is the mulberry molar, which appears in the first molars of the second dentition. The cusps of these molars are defective and dwarfed. The teeth are deficient in enamel, and decay is a frequent and early manifestation. These dental abnormalities occur entirely in patients with prenatal syphilis.

Other manifestations of prenatal syphilis present themselves on the face of the patient, and dental abnormalities may or may not be present coincidentally with these signs and symptoms. One of the most frequent complications of prenatal syphilis is an inflammatory clouding of the cornea of the eye known to syphilologists as interstitial keratitis. This condition is always bilateral unless involvement

of the uninvolved eye is prevented by treatment. Scarring and fissuring about the oral cavity, termed rhagades, is a relatively infrequent manifestation of congenital syphilis. In syphilis transmitted before birth, destruction of the nasal septum may lead in later life to so-called saddle deformity of the nose. Deafness is likewise a frequent manifestation of prenatal syphilis.

CASE FINDING

The dentist who suspects the presence of syphilis when he sees the manifestations described above can by referring his patients to a qualified physician do much toward the discovery of unknown syphilitic patients. In taking this action, he is helpful to the patient in insuring a more normal existence in the future, and he aids the community by rendering communicable syphilis noninfectious through treatment.

Health officers urge that all patients with syphilis of less than a few months' duration be questioned in order to obtain the names of contacts who may have been exposed to the disease. The examination of these contacts in order to determine whether the syphilitic infection has been transmitted is a most important contribution to syphilis control. Second only to it is the use of the routine serologic test by all physicians in an attempt to discover hidden syphilis. The dentist who learns to suspect syphilis will aid both health officers and physicians in the application of these two case finding methods.

CONTROL THROUGH TREATMENT

Once the syphilitic patient is discovered, the control plan calls for the administration of adequate treatment. Treatment through the use of modern antisyphilitic drugs, the arsphenamines and bismuth, is highly effective. The communicable lesions of syphilis heal after one or two doses of the arsphenamines and do not recur if an adequate treatment schedule is completed. The late crippling manifestations of syphilis are likewise prevented in the vast majority of patients when proper treatment is given in time.

The best results are attained in early syphilis when treatment is instituted during the first year of the disease and a minimum of thirty doses of one of the arsenicals and sixty of one of the bismuth preparations are administered. Eighty per cent of the patients may expect to recover. In the symptomless stage of syphilis, prevention of the late crippling manifestations is almost as effective when this treatment scheme is used.

Especially cheerful is the promise for the prevention of the prenatal transmission of syphilis when proper treatment is given to the expectant mother. If syphilis is diagnosed and treatment started before the fifth month of gestation, syphilis in the new-born child can be prevented in more than nine out of ten cases. Proper treatment in pregnancy consists of a minimum of ten doses each of one of the arsphenamines and one of the bismuth preparations. When no treatment is given the syphilitic expectant mother, the child is apparently nonsyphilitic in less than one case out of five.

SUMMARY

The dentist has an important part in the control of syphilis. Particular emphasis has herein been given to the following points:

1. The average dentist is called on to render professional service to one syphilitic person out of every twenty patients who consult him.
2. All patients who seek dental care and who are infected with syphilis are not necessarily capable of transmitting the disease. Fortunately, the number of patients with the communicable lesions of syphilis in the oral cavity represents only a fraction of the total number of patients.
3. The chancre and the mucous patch are the most common communicable lesions of early syphilis.

4. The recognition of the oral lesions of late syphilis is important.

5. The dentist's contribution to the problem of case finding may contribute to the syphilis-control campaign.

6. The control of both acquired and prenatal syphilis may be materially aided by the wary dentist who arranges for the administration of proper treatment of his patients by a physician.

THE BAKED PORCELAIN INLAY

By Paul K. Hill, D.D.S., Tucson, Ariz.

WHY the seeming neglect of the baked porcelain inlay? Why the misconception of most of the dental profession today that the baked porcelain inlay operation is a difficult procedure?

It is surprising to note, in reviewing past dental literature, how little has been written about the baked porcelain inlay. Volumes have been written on the porcelain jacket crown, and on the characteristics and manipulation of porcelain itself scarcely a detail has been omitted, but the porcelain inlay is only briefly mentioned. It is also amazing to note the statement that only 5 per cent of the dentists in practice today use the porcelain inlay in their operative procedure.

The conception that the porcelain inlay operation is difficult is a false one. Ability to master a refined technic in any branch of operative dentistry can be developed only by the expenditure of time and effort. The manipulation of porcelain in any form is not so difficult, but is merely detailed, and the details involved in fabricating a good porcelain inlay are no more than those necessary to produce a well-fitting gold inlay or to manipulate gold foil properly. It is true that there is nothing more discouraging than the many failures encountered by beginners in porcelain work; but who of us has not had many failures in gold inlay technics or in attempting the tedious process of manipulating gold foil? Almost any branch of operative dentistry is apt to take us along a rough and rugged road, and the porcelain inlay operation presents no more pitfalls than any of our other endeavors.

We are all aware of the fact that there is no more satisfactory restoration in dentistry than that produced by well-manipulated porcelain. There is no other material in dentistry that simulates the natural tooth as does porcelain. Porcelain has certain characteristics and qualities which make it an ideal material for replacing lost tooth structure. Esthetically, it is outstanding, and it is especially adapted to filling small cavities in anterior teeth. Even with a knowledge of this fact, many men have adopted substitutes or imitations in the form of plastic cements which slowly destroy tooth structure instead of replacing it permanently. A few years ago, a man well known in porcelain inlay work made this statement, "Many men who are using such imitations are slowly digging their professional graves with the agate spatula and the celluloid strip"; a fact well-known to many of us.

Regardless of the untiring efforts of the dental profession and the programs instituted by dental societies to educate the public as to its dental needs, the fact

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remains that the patient will always seek dental care for two reasons: pain and appearance. The loss of a single anterior tooth means far more to any patient than the loss of all masticatory function, because of the loss of facial harmony and personal appearance.

Speaking of esthetics in dentistry, Oliver Wendell Holmes said, "There is no pearl in any royal crown for which a young queen would give one of her front incisors."

Esthetics is defined as the theory or philosophy of perception; the science of the beautiful in nature and art. Esthetics in dentistry is therefore the science of harmony and beauty of the human mouth. The fact that many fastidious patients today are demanding more and more that dentists restore lost tooth structure with a substance which harmonizes in every respect with the natural tooth makes it necessary for us to develop our technic in the manipulation of dental porcelain.

The baked porcelain inlay procedure is not for the careless man. The work is not difficult, only detailed, and most of the difficulties that arise in the process of constructing a porcelain inlay can be eliminated by careful attention to details. Certain features, such as cavity preparation, color selection and contouring, require a degree of skill that can be acquired with a certain amount of effort. The results achieved after the difficulties have been surmounted are more than adequate to repay us for our efforts. Unfortunately, many practitioners are inclined to delegate a certain amount or all of their porcelain work to commercial laboratories. As we all know, this cannot be successfully done in the making of porcelain inlays because the individual touch is necessary to assure the perfect result.

ADVANTAGES AND DISADVANTAGES

The primary function of any filling material is the preservation and conservation of teeth. The baked porcelain inlay is a non-porous, hard, resistant filling which will answer all requirements. It is especially adapted to cervical cavities which extend under the free margin of the gum tissue, because there is no material to which the tissue takes more kindly than to properly fused porcelain. It is also particularly adaptable in Class III cavities because of its density and hardness. It restores and maintains contact and the interproximal relationship, features that plastic imitations do not have. Strange as it may seem, recurrent caries is reduced to a minimum around the baked porcelain inlay; which is difficult to explain, unless it is due to the fact that the bacterial plaque does not adhere readily to baked porcelain.

The advantages of using the porcelain inlay as a filling material are: 1. It is much more harmonious with the natural teeth. 2. It is a poor conductor in thermal changes and a non-conductor of electricity; hence, it lessens pulp irritation. 3. The margins of cavities filled with porcelain are not easily attacked by caries. 4. It is comparatively easy of introduction. 5. It is more consistent with the natural tooth structure than metallic fillings are. 6. It resists the accumulation of food débris and plaques.

Among the disadvantages of using porcelain are the following: 1. It is friable and fractures easily if there is not sufficient bulk to insure strength. 2. Matching of colors is difficult. 3. The use of cement as a retaining medium may cause change of color of the inlay.

INDICATIONS AND CONTRAINDICATIONS

Porcelain inlays are indicated in all classes of cavities found in the anterior teeth and in all Class V cavities located in any part of the mouth; that is, all gingival third cavities. The gingival third of all teeth is perhaps the commonest location of the porcelain inlay because the free margin of the gingiva takes very kindly to highly fused porcelain. Care should be taken, in building a Class V

porcelain inlay, to maintain gingival contour. In many instances, it is advisable to "overcontour" the inlay in this area in order to maintain gum tension. It is also adaptable to this locality because it resists the formation of plaques, the recurrence of caries being thereby reduced.

The porcelain inlay is sometimes used in Class I cavities in lower molars and bicuspids, especially where metal fillings would be conspicuous.

It is also used in a box-like cavity preparation on the mesial surface of upper and lower bicuspids when the patient is averse to any metal showing. This type of inlay is recommended with certain restrictions because of occlusal stresses brought to bear in these areas.

In restoring incisal angles, care must always be exercised not to depend too much on the strength of the porcelain to withstand incisal stress unless sufficient bulk can be obtained. It is often necessary to reinforce this type of inlay with a hard gold inlay backing.

In cases occasioned by automobile accidents and other circumstances, of fracture of enamel corners in which the dentin has been left intact, a quarter or half jacket preparation may be made. In this preparation, the gingival seat and incisal portion of the dentin are inclined toward the center of the tooth in order to resist stress and dislodgment. This type of preparation allows for conservation of tooth structure and restoration of esthetic appearance.

COLOR

Matching of colors in porcelain inlays, a somewhat difficult problem, is made easier by an understanding of the colors in natural teeth and the colors of porcelain as given to us by the manufacturer. Comparatively few colors of porcelain are necessary in the making of porcelain inlays as we are dealing principally with the two primary colors found in teeth; namely, yellow and gray. The color problem is one which has to be worked out by the operator himself. It can be done by selecting the colors mentioned and increasing or decreasing the depth of hue, brilliance and saturation by adding to the yellow or gray, white, pink or brownish gray.

Proper cavity preparation will also lessen the color problem and render the inlay less conspicuous.

Cementation affects the color of inlays, but since the advent of certain silicate cements, this difficulty has been materially reduced.

Frequently a cervical inlay is placed in a cavity without cement and the color is perfect. After cementation, the color is altered. If a colorless cement were available, the color problem would be solved; but such is not the case: all cements have color and are more or less opaque. This condition is most frequently remedied by the selection of cement one shade darker than the tooth. The technic of cementing porcelain inlays is one which must be developed by the operator himself, because color of teeth will vary, but, with a little practice, it can be easily mastered.

SEPARATION

In proximal cavities in the anterior teeth, in order to gain access for cavity preparation and impression taking and to conserve tooth structure, separation of the teeth is necessary. This is especially necessary in the preparation of a Class III cavity. It may be accomplished either by the slow separation method or by means of a mechanical separator. The slow method of separation is preferred. Mechanical separators are more often than not cumbersome and interfere with cavity preparation and especially with obtaining impressions of the cavity.

In the preparation of cavities for porcelain inlays, the principles laid down by G. V. Black are observed and followed closely. The marginal outlines of the cavities should be devoid of sharp angles. All lines should be gently curved; which allows for better adaptation of matrices

and for better fitting margins. The internal part of the cavity is cut in the form of a box with a flat or convex seat and parallel or slightly divergent walls, which act as the principal means of retention.

The Class V cavity is the simple box type of cavity, the marginal outline being in the form of gentle curves following the contours of the gingivae, and the cavity margin incisally being cut in a gentle curve as it extends mesiodistally.

Wherever it is possible, the box form of cavity is used in the Class III preparation. Owing to the position of the cavity in the tooth after the box is established, the labial wall is cut back or beveled so that it is perpendicular to the labial surface of the tooth. This places the cement line of the finished inlay exactly edgewise and renders it inconspicuous. This so-called bevel, which may also be considered a convenience bevel, is sometimes used on the lingual wall when access to the cavity is from the lingual aspect and the withdrawal of the impression in that direction is desired. In certain cases wherein the lingual wall has been undermined by extensive caries and must be destroyed, some operators advocate complete cutting away of this wall, leaving only the three-walled cavity, and advocate withdrawal of the impression from the lingual aspect. This deprives the operator of the fourth wall of retention, which can be obtained by preserving a part of the lingual wall and utilizing a cut into the dentin both linguo-incisally and linguogingivally. This affords the more typical form of Class III cavity and provides the fourth wall of retention.

In Class IV cavities, the box form of retention is frequently used where the gold backing is not necessary. This makes a very satisfactory porcelain inlay and, where bulk can be obtained, a strong and resistant restoration.

Both the Class III and Class V cavities are outlined by means of an inverted cone bur of medium size, with the right angle bur in the straight handpiece, which allows for more control because of the short shank. After the cavities are outlined, they are cut and completed with sharp cutting instruments of the smaller sizes, principally the angle forming type of instrument such as is recommended by W. I. Ferrier in gold foil work.

INDIRECT TECHNIC

There are many methods of producing a porcelain inlay and all seem to have equally good results. Some operators prefer the direct method, some the indirect method, and still others the direct-indirect method. This is discretionary with the operator and can be worked out according to his own liking. The direct method, where it can be used conveniently, assures an exceptional result, as also does the direct-indirect method. However, with the advent of the electroplating process of making dies over wax impressions, results seem to be as exact as those of other methods. This indirect technic eliminates long and tiring sittings for the patient and reduces chair time to a minimum for the operator.

IMPRESSION TECHNIC

The impression of the inlay cavity after it is prepared is perhaps the most important step in the making of a porcelain inlay. An absolute and perfect likeness of the cavity must be produced, and this can be done only with a perfect impression.

The technic of impression taking for different types of cavities is determined in part by the position of and the access to these cavities. Wax under pressure is used for all impressions, the pressure being necessary to force the wax into the sharp lines and point angles of the cavity to give the most minute detail. The ultimate impression of all cavities is made in a soft wax. This wax is three-fourths electroplating wax and one-fourth No. 5 black wax, melted together, which we will call, for lack of a better name, the "gray wax." This wax has a dead, soft quality which allows it to be forced into the confines of

the cavity. It reproduces all details perfectly and has the peculiar property of having no spring, which minimizes distortion. The No. 5 black wax is added to the electroplating wax in order to give it bulk and tension, a property which the latter does not seem to have, it being more or less flaky and granular after heating and cooling. This wax is also unexcelled as a recipient of the bronze powder and the initial deposit of copper in the electroplating process.

TAKING AN IMPRESSION

In order to take the impression of a Class V cavity, a tray must be made. A small piece of brass is cut and contoured to slightly overlap all margins of the cavity and so that it will raise the gum tissue when pressed beneath it. This tray is reinforced with soft solder so that it will resist pressure without distortion. A small cone of hard inlay wax or compound is placed in the tray, and the end of the cone is softened and forced into the cavity. This wax is chilled and removed with no particular attention to the detail desired. Over this impression, with a pair of fine-nosed wax pliers, a film of the gray wax is flowed, passed hurriedly through the flame, pressed firmly into the cavity and chilled under pressure. The compound or inlay wax, owing to its density, acts as a plunger which forces the gray wax into the angles of the cavity, affording a perfectly detailed impression. This impression wax is removed from the cavity and examined for detail, and if it is not as desired, the operation of rebasing is repeated until the impression is satisfactory.

The tray employed for the impression of the Class III cavity is a small piece of 1/1000 platinum foil. A strip of platinum foil is passed through the space which is made for the cavity preparation and the cavity margins are outlined on the platinum foil by means of a thin burnisher. This foil is removed and a tray cut so that it will overlap the margins of the cavity to the extent of 1.5 to 2 mm. With a ball burnisher, this tray is cupped. A thin film of sticky wax is flowed on the foil and this is covered with a layer of gray wax. The cavity is now dried and lubricated with expressed oil of almond, which prevents the wax from sticking to the cavity when it is forced to place. A cone of the gray wax is now softened, placed in the cavity and pulled into place with a previously oiled celluloid strip, held under pressure and chilled. The margins of this pattern are carved flush with the cavity, and the pattern is removed and examined for cavosurface detail. This primary pattern is replaced in the cavity.

The previously waxed platinum tray is now placed over the primary pattern and settled against the tooth with a warm instrument. A small piece of rubber dam is inserted in the interproximal space and drawn tight over the platinum tray, care being observed not to allow the tray to slide either labially or lingually. The tray may be guided to place, being gently warmed through the rubber dam with a warm instrument, the operator making sure that all margins are overlapped. The instrument is again warmed and the tray pressed or burnished on all margins of the cavity. The warmed instrument softens the two waxes and causes them to unite. The wax is chilled with the rubber dam still held taut. The rubber dam is removed and ice water is again flowed over the impression. The impression is gently teased out of the cavity and removed through the separated space, and is immediately mounted in a pat of soft plaster so that it can be handled without distortion when the bronze powder is applied for electroplating.

The same technic can be employed in taking a Class IV impression when the box form of cavity is used. If the box form is not used and it is possible to use a band in taking the impression, a hard inlay wax is employed for the primary impression and this is rebased with gray wax.

After the die is completed and mounted on a swaging ring, a platinum matrix of three four-thousandths inch thickness is burnished.

BURNISHING THE PLATINUM MATRIX

The burnishing technic is as follows: A piece of platinum considerably larger than the cavity is cut and placed over the model and, with finger pressure, the cavity form is outlined. With a small ball burnisher in a spinning motion, the platinum is carried down into the cavity until it becomes taut. With a sharp instrument, the matrix is cut in the form of a cross. The cut having relieved the tension on the platinum, it is now pressed and burnished into the angles in the bottom of the cavity, care being observed not to tear the cuts to the margin or to allow any wrinkles to develop. This piece is now swaged in a swager with a rubber plunger. A small piece of platinum of the same thickness is now placed over the cavity, and, with finger pressure, the cavity outline is again obtained. With sharp scissors, the platinum is trimmed inside the outline of the cavity and this piece is pressed into the bottom of the cavity with a piece of spunk and burnisher forming a pattern of the floor of the cavity. This unites the four corners of the platinum which have been spread in the bottom of the cavity by the previous burnishing. This is also swaged to place. The matrix is removed and the two pieces of platinum are sweated or welded together, in the flame of a blow torch. The matrix is carried to the die and waged again; which completes the burnishing process. The technic of burnishing platinum matrices is that of A. J. Brock, Portland, Ore.

BAKING

The first layer of porcelain is usually placed in the matrix, held in a pair of lock pliers out of the die. Only a thin layer is applied and this is vibrated to place so that it will cover or bridge over any small holes that may be present in the matrix. This prevents porcelain running under the matrix as is the case when it is left in the die. The porcelain is carried into the muffle and fired to a high biscuit finish. The matrix is carried to the model and swaged, and the second application of porcelain is made, the operator at this time filling the matrix full and ditching at the margins to prevent pulling of the matrix while firing. In large inlays, in which a great bulk of porcelain is required, on the second bake, or when the cavity may be unusually long or curving around the surface of a tooth, the porcelain is crisscrossed in order to control warpage of the matrix. This is fired again to a high biscuit finish and placed in the die and swaged. At this point, the matrix is spun over the cavity margins with a flat burnisher and the third application of porcelain is made, filling up the ditch around the margin or filling in the crisscross if such has been made. In large inlays, after this bake, the porcelain is again crisscrossed or ditched. In the case of a small inlay, the margin is barely relieved, a small moist sable-hair brush being used. This is again fired to a high biscuit finish, the inlay is placed in the die and swaged, and the matrix is again spun over the margin. The final application of porcelain is now made, being carried completely up to the margins and allowed to remain there. The inlay is carried to the furnace and fired to complete vitrification or glaze.

I prefer to use medium fusing porcelain in the making of porcelain inlays. Most inlays can be completed in four bakes. Each bake is carried to 2150° F. in a properly calibrated muffle for one minute on the first three bakes, the final bake being held at this temperature for three minutes, which gives completely vitrified porcelain with a high glaze.

The application of each layer of porcelain after each bake is greatly facilitated by wetting the surface of the inlay. This allows the porcelain to flow up to the margins and fill in all crevices which

may have arisen from shrinkage. It also minimizes the formation of air bubbles and entrapping of any free air on the surface of the previously baked porcelain. After each bake, the porcelain is thoroughly vibrated and condensed, and, by proper firing, this technic gives a very dense, non-porous, highly glazed inlay which answers all requirements.

CEMENTING AND FINISHING

The matrix is now peeled from the inlay by grasping the inlay between the thumb and forefinger and gently rotating the platinum from the inlay in a pulling motion with fine-nosed pliers, care being taken not to touch any of the margins with the pliers, to avoid chipping or fracture. The matrix can be peeled off much more easily if the inlay is previously moistened with water.

The inlay is now tried in the cavity and examined for color. If margins, contour and color are correct, the cavosurface of the inlay is etched with a fine knife-edge stone and a groove is cut around the cavity wall of the inlay. A corresponding groove should be cut in the cavity of the tooth with the point of a sharp cutting instrument or a very small inverted-cone bur. This groove acts as a lock or extra retentive medium, which is particularly essential when silicate cements are used for cementing porcelain inlays.

The cavity is now sterilized, dried and varnished, and a suitable mix of silicate cement is made and introduced into the cavity. The inlay is picked up on an instrument on which a small amount of cement is allowed to remain, carried to the cavity and pressed firmly to place, slight but firm pressure being maintained with the point of an orangewood stick until the cement is set. Silicate cement should be treated with the same precautions as are used in handling synthetic porcelain. It should be kept dry until set and moist after setting. After the cement is set enough that the excess may be removed, this is chipped or broken away and a varnish applied. At a subsequent sitting, the margins are examined, and if there are any slight overhangs, these may be polished down with any one of the rubber porcelain polishing wheels or fine disks. It is sometimes necessary to go over these inlays with polishing wheels or disks to eliminate the extra high glaze or glassy appearance so that the inlay will more nearly harmonize with the natural tooth.

CONCLUSION

Effort and interest in any branch of operative dentistry tends to improve one's ability in all branches of dentistry. This is especially true of interest in porcelain inlays, which is due, no doubt, to the especial attention to detail necessary for success. It is true that the technic of making a well-fitting porcelain inlay may be tedious, but, as in any other branch of the porcelain art, there is nothing more gratifying to the operator or patient than a well-executed or finished piece of porcelain. The knowledge of handling porcelain renders us capable of combining service with esthetics.

801 Valley National Building.

A CURATIVE TREATMENT FOR SUBLUXATION OF THE TEMPOROMANDIBULAR JOINT OR OF ANY JOINT*

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SUBLUXATION of the temporomandibular joint is, comparatively speaking, of frequent occurrence and to date its treatment has been very unsatisfactory. To safeguard such an affected joint for one whole year against a subluxation is well nigh impossible. Yet this treatment is the best one known today to afford relief. However, I submit a new treatment which restores the normal function of this joint quickly, effectively and with a very high percentage of cures.

Up to the present, the pain, the clicking, the subluxation and even the dislocation of some joints in the body had to be endured. This is no longer necessary; for, under the treatment that I have developed, all the above-mentioned symptoms disappear.

As I have said, the best treatment known to date is rest. Surgical operations have been tried with some success and physiotherapy has been employed in an effort to help these patients.

Various other means of treatment have been introduced to relieve this annoying ailment. Among them may be mentioned orthodontic and prosthetic devices and pressure pads applied in front of the ears and held in place for several months by a firm steel spring passing over the calvarium, as recommended by Riley.[1] Devices of similar character were suggested by Brophy.[2] Wiring the jaws in occlusion, plus bandaging, continued for six months or longer, has also been used. Costen and George[3] altered the bite, with good results in some cases.

Several years ago, when injections came into vogue for the treatment of hernia, injections of alcohol and tincture of iodine were used in subluxation of the jaws. These caused severe pain, requiring a local anesthetic for alleviation. The results were poor, the distress was very severe and the dangers were great; hence, it was soon abandoned. Then, too, alcohol injections in the treatment of hernia have had very serious effects according to Goldhahn[4] and Jacobi.[5]

In the treatment of subluxation of the jaws, Morris[6] limited motion with mattress sutures inserted laterally through the capsular ligament. The removal of the meniscus has had good results, as reported by Gernes,[7] Combier and Murard,[8] Wakeley,[9] Moczar[10] and others.

Recently, much clinical and experimental work has been done with sclerosing agents, by McDonald,[11] Pina Mestre,[12] Mayer,[13] Wyss,[14] LaRochelle,[15] Batrud[16] and Rice.[17] All these investigations were carried on for the purpose of devising a successful treatment for injection in hernia. Alcohol and tincture of iodine have been mentioned. Sodium morrhuate, potassium oleate, quinine and urea hydrochloride and hypertonic saline solution are among the simpler agents so

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used, but other solutions, of the so-called "shot gun" character, were also introduced. An example of the latter is furnished in the solution used by Zollinger[18] which consists of alcohol, chloroform, acetic acid and ferric chloride. Rice[19] made his initial experiment with the thuja solution. He warns that doses larger than 5 minims will surely cause a slough. He also experimented with a tannic acid preparation which, when introduced intravenously, produced symptoms of extreme toxicity. Substituting sodium psylliate, he found that there was no effect shown either immediately or at necropsy.

The work done on sclerosing agents, as noted so far, was an effort to find such an agent suitable for use in curing hernia by injection. My interest in these agents centered not on the cure of hernia, but on the cure of subluxation or luxation of the joints, notably the temporomandibular joints. This required a critical study of the effects of all these agents when applied to diseased joints and to normal joints. The conclusion arrived at by this study is that a 5 per cent sodium psylliate solution is the ideal agent for such therapy.

In my investigation, I found that the action of sodium psylliate produces a firmer gross specimen in all infected areas than does that of sodium morrhuate. Also, it causes a considerably larger amount of uniform collagenous connective tissue to develop. Nearly 2,300 celloidin sections were made in the microscopic phase of this investigation. A study of the joints injected with a 5 per cent sodium psylliate solution revealed a thick fibrous capsule. Injections up to six in number into the temporomandibular joints of dogs caused practically no loss in the opening between the incisor teeth, and at necropsy normal joint cavities with firm fibrous capsules were revealed.

Other experiments made on dogs revealed large areas of firm fibrosis in from two to three weeks after massive subcutaneous injections, and without deleterious effect. In similar experiments, this drug did not act on sensory nerves as far as the mandibular and infra-orbital nerves are concerned, nor did its use cause paralysis on injection about the femoral or sciatic nerves. This solution, when injected into the peritoneal cavity, had no immediate or remote effect. Similarly, there was no effect from injections into the lung, liver, gallbladder, blood vessels or heart.

Hence, the following conclusions relative to injections with sodium psylliate seem justified: 1. It is the best of the sclerosing agents in the treatment of the type of joints with which this paper deals. 2. A dense fibrous capsule is formed when this solution is injected into joints, without any effect on the joint cavity but restoration of joint function. 3. There are no adhesions in serous cavities following its use, and no deleterious effect on the viscera need be feared. In the dog, the toxic dose is estimated at 6 cc. to each kilogram of body weight.

EXPERIMENTAL WORK

My animal experiments showed: (1) no alteration of the joint cavity, the fibrosis occurring in the ligaments; (2) no change in the ligaments other than thickening, and thus strengthening, of the ligaments to hold the joint within its cavity; (3) no symptoms from injection of large doses directly into the bloodstream; (4) no symptoms from injection into the heart; (5) little discomfort from injections into the joint cavity, and (6) no infection following treatment.

A detailed comparison of the effects of the other solutions suggested with the agent I chose is not possible here. However, I shall note the effects of a few of those subjected to careful study in this work. All fell short of the ideal, not only those here mentioned, but also all sclerosing solutions, recommended by various authors and subjected to critical study.

The harmlessness of this treatment and the quick results obtained should make

this the treatment of choice. In one week, a definite improvement is noted, the degree depending on the amount of relaxation of the tissues before treatment is started. The treatment consists of injection into the joint of 0.5 cc. of a solution of 5 per cent sodium psylliate.

Naturally, no such injection is made until a complete history and examination of the joint relative to its degree of relaxation and other peculiarities, and a careful clinical examination of the external auditory meatus, drum head, etc., show that such treatment is indicated.

TECHNIC

In making the injection in such cases, the ball of the index finger is placed in front of the tragus and the patient is instructed to open the mouth wide enough to cause the head of the condyle to subluxate, or to produce the "click" or abnormal movement of the fibrocartilaginous disk. The needle is inserted in the joint cavity and from 0.25 to 0.5 cc. of the solution is deposited inside the capsule. This is allowed to diffuse through the joint, producing a uniform fibrosis around it. The injections are repeated weekly or biweekly in both joints until there is sufficient fibrosis. The desired amount is obtained in from three to five weeks. The injections therefore usually number from two to four at intervals as stated above.

This technic should not produce any more disturbance than the pricking sensation of the needle and a slight feeling of fulness at the time of the injection. If pain is encountered from twenty to thirty minutes later, the patient may be made comfortable, if necessary, by the administration of a sedative or anodyne and the application of heat to the parts involved.

The operator should remember that in this region there are many important structures, such as the internal maxillary and the internal carotid arteries, the middle and the internal ear, the brain, parotid gland and facial nerve. Therefore, this joint should be injected with proper caution.

That the general practitioner may afford relief to many people suffering from loose sacro-iliac joints or other troublesome joints is my hope. The industrial as well as the orthopedic surgeon will, I am sure, welcome the quick response to this treatment in patients afflicted with acute traumatic or chronic sprains, leaving joint capsules in a weakened condition.

The apparent specific non-injurious fibrosis developed by the use of this solution should open a field in the practice of medicine and surgery for the treatment and cure of many afflictions in which fibrosis is desirable.

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CONTROLLING DENTAL CARIES

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WHEN we contemplate the multitude of diseases with which the physician must deal; and realize that the causes of a great majority are definitely known; that preventive measures are successfully practiced; that some of these diseases are not only being prevented, but also give promise of being completely wiped out, it appears somewhat of an indictment against our profession that, with so few diseases to combat, so little is known of their causes and the knowledge of prevention is almost at the zero point.

The diseases with which the two professions are concerned differ widely. The physician's problems more often deal with the most tragic of human experiences—unlimited suffering and death—while, in the public mind, the worst that can come from dental diseases are toothache and the necessity of wearing artificial teeth.

It is well understood by both the dental and the medical profession, and to a limited degree by the laity, that the sequelae of dental disease may, and often do, reach far beyond the oral cavity and cause grave and serious complications.

It was anticipated by many members of the dental profession that this knowledge would have a salutary effect on their efforts at preventing diseases and saving teeth. Strangely enough, it has had quite the opposite effect. The physician, seeing an opportunity to lay the cause of many of his not-too-well understood diseases to teeth and the dentist, follows the line of least resistance and reasons, "Eventually, why not now?" The suffering patient, seeing in each tooth a potential danger to his general health, submits to the consensus of opinion and joins the rapidly increasing army of the edentulous. Led by the exodontist and followed by the prosthodontist, a new era in dentistry has been ushered in.

All this has resulted in a letting down in the methods for saving teeth. The few have gone ahead in improving their operative skill; the many have either made no improvement or lapsed into in-

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different, careless habits. The few are giving more thorough and intelligent attention to the investing tissues; the many pass by without giving a thought to gingivitis and incipient pyorrhea. When they do note signs of it, it is well advanced, and they advise the patient to have his teeth out.

Dentistry could well moderate, for a time at least, its efforts to develop a multiplicity of mechanical contrivances for replacing teeth, and devote some concentrated thought to methods for saving teeth. It is not a good omen to see our profession drifting back from science to mechanics.

To save teeth requires not only a high degree of mechanical skill, but also knowledge of the basic sciences. The well-balanced dentist is the man well grounded in both.

To run to extremes seems to be an inherent characteristic of the human mind. To do means too often to overdo.

When Black came forward with the theory of "extension for prevention," dentists everywhere seized on the principle at once, only to carry it to extremes.

I came into the profession at the height of the pulp-destroying era. At one of the first meetings of the then National Dental Association that I attended, I heard a statement by a prominent man that I shall always remember: "If a pulp gets in my way, I remove it."

We see today in surgical clinics, moving picture films, etc., radical measures which could well be classified as practical applications to the foregoing statement. Well might these operators say, "If a tooth gets in my way, I remove it." Indeed, we are in the midst of a tooth removing orgy. When the history of dentistry is written, this will be known as the era of radical, ruthless tooth extraction.

Is that dentistry's goal? Are we or are we not members of the healing art? Does our contribution to humanity consist of artificial substitutes? Has our place in the sun been filled with glass eyes and wooden legs? If this is all, we deserve the slurs and darts aimed at us every day by a ridiculing public.

Have we forgotten the old adage about "an ounce of prevention"? Do we forget that "a tooth in the jaw is worth five in the artificial denture"? Do we forget the prominent rôle teeth play in the beauty and harmony of the face? Do we realize how miserably, after all, our best efforts

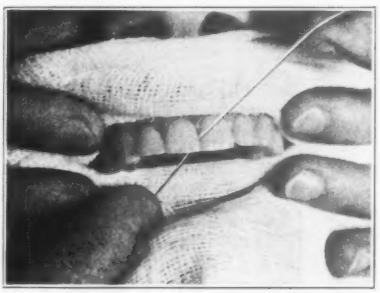

Fig. 1.—Method of application to proximal surfaces of incisor teeth when rubber dam is not used. It is better, however, to use the dam on patients past ten years of age. Very good results may be secured by using napkins as shown above, provided the surfaces are thoroughly dried with compressed air. If some of the solution gets on the septal tissues, no injury results.

Fig. 2.—Method of removing stains from labial surfaces using triple X silex and rubber cups. This should be done immediately after each treatment.

fail of imitating the natural organs? The man who thinks that he has perfectly imitated the natural teeth ofttimes deceives no one but himself.

In fairness, we must agree that ours is the only profession capable of replacing a lost human organ with a substitute which not only closely resembles that which was lost, but, what is more, functions. Glass eyes do not see nor do artificial fingers play the piano. I have often thought that if we had no substitutes, or if our substitutes were less beautiful and less efficient functionally, a greater value would be placed on the natural dental organs and the patient would cooperate better in our efforts at saving them.

The loss of the teeth is little short of a tragedy. The sickness, the suffering, the lowered efficiency, the loss of time and the dentist's fees constitute a formidable economic burden.

To combat this calamitous result of the loss of the teeth, the dental profession came into being. How we are succeeding is evidenced by the millions upon millions of open cavities in the mouths of little children and the sickening dental specters seen through the parted lips of the man on the street. Knowing all this, can we keep our professional pride and hold up our heads in the face of defeat? Can we justify ourselves by laying the responsibility at the door of the parent, even though some of it belongs there? One thing is sure: The condition of the child's mouth cannot be laid on the child! In some way, aid must be found for this child. Where can he go except to the profession trained and qualified to serve him? If we are trained and qualified to serve, why has he not been served? The answer is found in the fact that we have not been capable of handling the situation. The sooner we see and admit this, the better.

The facts point definitely to the conclusion that our present methods of attack are wrong. We had just as well attempt to keep back the waves of the Atlantic Ocean with a broom as to attempt to control dental diseases with present methods. Notwithstanding the great advances in all lines, the task is constantly getting farther beyond us. Each improvement carries with it added effort and time, with each an additional cost to the patient. From the very nature of the problem, all

Fig. 3.—Layer of sclerotic dentin, a wise provision on the part of Nature to protect the pulp. With the treatment advocated by the author, caries is arrested and the pulp is given time to form this protecting layer of hardened tissue.

Fig. 4.—Molar treated in the laboratory. The dotted line shows cavity outline. The black metal-like tissue was carious before treatment. Repeated treatments give this result. The stain has been removed with chisel or bur from all walls except that over the pulp, which is allowed to remain. This prevents shadows after the filling is made. Notice the highlights from the burnishing. In this respect the tissue resembles copper amalgam.

restorative methods fail to measure up to the requirements.

The solution, if one is ever found, will not be along the present lines of procedure. It lies in the slogan of this convention; that is, prevention. Though our knowledge of prevention is vague and lacking in scientific background, from the little we have may spring great and ever-broadening accomplishments.

PREVENTIVE METHODS

As one of the possible approaches to the control of proximal caries, I bring once more to your attention my results in the use of Howe's ammoniacal silver nitrate.

To begin with, we should modify some of our ideas as to what constitutes incipient proximal caries.

Fig. 5.—Tooth which was treated in the mouth. It was uncomfortable and began aching, and so was removed. On section, it was found that the solution had penetrated as far as the caries extended. All the tissue under the white line was black and hard as ebony. The broach shows the exposure which caused the trouble. This degree of penetration is impossible if the eugenol is applied first.

The idea is, I think, generally entertained that "the first sign of the beginning of caries on the enamel surface is an unnatural opaqueness caused by the interference with the refraction of light rays, owing to the irregular dissolution of the calcium salts of the enamel." This is not the beginning of caries, but is the result of caries active over an extended period of time.

Caries results from the presence of acid-forming bacteria. They are always microscopic; so also are the first results of the activity on the enamel surface. This first etching is never visible until the process is well advanced. It progresses for an extended period of time before there are any visible signs of its presence. Its presence later on may be detected by the unnatural opaqueness mentioned above, or it may be made visible by a pigmentation peculiar to slow progressing caries, or loss of surface continuity.

H. E. Friesell came near to stating the condition concisely when he said, "Caries is a slow process and may progress microscopically for a considerable period of time before definite injury to the tooth structure can be recognized." I would take exception to the words "may progress" and substitute "always progresses."

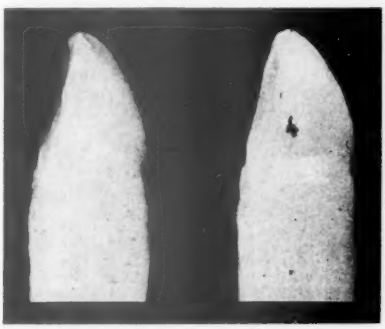

Fig. 6.—Freshly extracted tooth which gave no evidence of proximal caries until the solution was used.

The idea that proximal caries does not begin until there has been a loss of septal gum tissue is an error, as is at once evident when we know that proximal caries incidence is greatest in youth, when the septal tissue is *in situ* and in more nearly perfect condition than at any other period in life. Proximal caries begins in youth and the septal tissue offers but little, if any, deter-

rent action. Proximal caries begins gingivally to the contact point, almost never occlusally or incisally to this point.

How do we know this? Because it is here that the first etching, the first opaqueness, the first cavity develops.

Let us start with a common premise and, provided it is a correct premise, we can arrive at a correct conclusion:

1. Proximal caries attacks a smooth, sound surface.

2. Invisible organisms colonize in a rather restricted area of this surface.

3. After a time, they generate an *invisible* acid of caries.

4. After an indefinite time, this acid of caries attacks the surface of the enamel and produces an *invisible* etching of this surface.

5. If this *invisible* colony remains undisturbed, this *invisible* acid of caries continues progressively more and more to deepen this *invisible* etching until there is loss of surface continuity.

6. All this progressed invisibly, up to this point (and this process may cover a period of years) before it could, until recently, be detected by any means known to dentistry. (Figs. 6-12.)

The logical approach to prevention would be, most assuredly, to eliminate the causative organism, be it one or many. With our present knowledge, we are unable to do this.

Not being able to do this, the next most logical approach is to interfere with the initial attack, before any injury to the surface has resulted.

One of the first efforts recorded along this line was vigorous polishing of the entire crown at very frequent intervals. Another was the slicing off of proximal surfaces with a stone, removing all contact points. Both these methods failed.

I became discouraged with our methods of examination. I tired of looking for cavities and, when at last finding them, filling them, frequently at a time when much damage had resulted to the tooth crown.

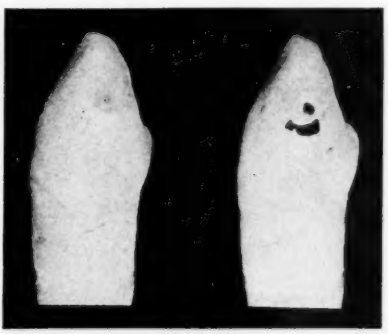

Fig. 7.—Cuspid which had, apparently, a perfectly sound proximal surface. The solution shows etchings which, in a short time, would have been a carious cavity.

Fig. 8.—Bicuspid demonstrating the fact that where there is no etching no stains will result. This surface was thoroughly treated and it is evident that no stain resulted.

Determined to find a method which would bring these invisible incipient etchings within the limits of my vision, I began experimenting with various disclosing solutions. These proved worthless, for in a few hours the solutions had faded out. I then tried Howe's ammoniacal silver nitrate, which I had found invaluable through years of experience in treating deep-seated caries. I soon discovered that it disclosed what I could not otherwise see. I had at last found a disclosing solution faithful in every instance in trans-

forming a microscopic etching into one easily seen with my unaided eye.

Satisfied with this result, I proceeded to apply the solution to large numbers of proximal surfaces of my patients' teeth.

A field unknown to me opened before my eyes. Little did I realize the amount of invisible, incipient caries on the proximal surfaces of my patients' teeth. I could hardly believe my eyes. I went to my laboratory and dried wet extracted teeth which showed no evidence of caries, and applied the solution to the entire crown. After I polished the stain off the sound enamel, I found that most of the proximal surfaces had etchings which I could not see until I applied the solution. (Figs. 6, 7, 8, 9 and 11.)

Fig. 9.—Cross-section of central incisor showing penetration in cavity on one side and surface etching on the other side. The etching is on the *surface only*. The picture makes it appear below the surface.

At that time, I supposed, naturally, that each stained spot meant a filling later. Time went by and, when opportunity permitted, we recalled the patients for fillings. Then came the still greater surprise in that no fillings were necessary, except where definite damage had been done before the solution was applied. Even many of these carious processes have been so effectually arrested as not to require fillings in over five years. (Fig. 12.)

It was not possible, because of the time required, to make systematic regular applications. The solution has been applied intermittently to the proximal surfaces of teeth for both young and old. Notwithstanding the lack of systematic treatments, some having been treated once a year, some four or more times, the results are surprising. In one case, silicate fillings had to be replaced in a mouth, but the caries has remained arrested over a period of seven years after application of Howe's solution. (Fig. 12.) During seven years of using the solution for this purpose, we have applied it on increasing numbers of surfaces of teeth until we have lost count.

If decalcification has progressed sufficiently to show in the roentgenogram, prospects for successfully arresting or stopping the process with this solution are

Fig. 10.—Molar treated in the laboratory and later sectioned. Arrow points to wall of thin, hard dentin surrounding the pulp chamber, which, as is evident, prevented the solution from penetrating to the pulp chamber. The pulp chamber is plainly visible.

not good. It is advisable, however, to apply it, for only good can result. But it is well to remember that fillings may eventually be required.

W. D. Miller perhaps made the greatest contribution to dental caries research of all time. As is well known, Dr. Miller investigated the results of using silver nitrate as a "preventive." He reported his findings before the British Dental Association and they were later published in *Dental Cosmos* in August 1905.

Because of his monumental contributions to dental research, his report must be given careful consideration. I can make only brief reference to it here.

For comparison permit me to say:

His work was done on ivory blocks and extracted teeth in the laboratory.

Ours was done on large numbers of teeth *in vivo*.

His work was done with inorganic acids never associated with caries.

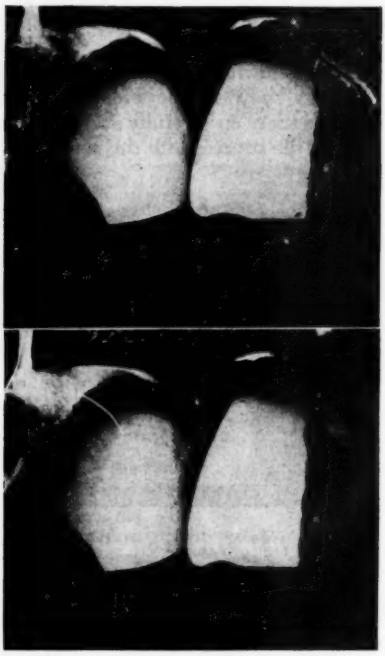

Fig. 11.—Proximal caries (in a boy, aged eighteen), a condition that is almost universal. The father of the patient was a prominent dentist and he gave assurance that there was no caries in the boy's mouth because he "polished all the surfaces every three months." The central incisors were separated and a picture was taken of the proximal surface, as shown above. It appeared free from caries. While the separator was in place, the solution was applied and reduced, definite etching being disclosed, as shown in the lower picture.

Ours is based on macroscopic observations in the presence of the acid of caries.

His was done on a half-dozen teeth far removed from the complex phenomena of oral dental caries.

Ours was done on thousands of teeth in mouths of highly susceptible people.

His control conditions were not analogous to oral conditions.

We have stopped incipient caries "in its tracks"; whereas untreated surfaces in the same mouth have rapidly decayed. (Fig. 12.)

His report was based upon results of one treatment.

Ours is predicated on repeated applications.

He used straight silver nitrate solutions which have a pH below 7 (acid).

We use ammoniacal silver nitrate with a pH of 8.5 to 9.5 (alkaline), which may or may not be a factor in surface treatment. (We do know, however, that the pH is of great importance in the treatment of deep-seated caries.)

Fig. 12.—Case presented seven years ago with cavities in the mesial surfaces of the right lateral incisor and the distal surface of the right central incisor. The author filled them with silicate cement. All other proximal surfaces were rather deeply etched as might be expected. These etchings have been "held in their tracks" for seven years in which time it has been necessary to renew the silicate fillings. *Untreated* proximal surfaces in the bicuspids and molars have, during this time, required fillings. The teeth were dried when this picture was taken. When they were flooded with saliva, the stain on the mesial surface of the left lateral incisor was only faintly visible. If the patient objects to these stains, which occasionally he does, they may be removed with a thin, fine grit strip.

His report took no account of the invisible surface etchings of proximal caries.

Ours is based on intervention at this stage.

It is well to remember that Dr. Miller conducted his study thirty-two years ago. Great advances have been made along all lines in the profession since that time.

When these facts are considered, we need not be surprised that Dr. Miller closed his study of this subject by concluding that silver nitrate is of questionable value for prophylaxis.

The laboratory and the microscope are indispensable in the pursuit of knowledge. The mouth must remain the final arbiter, the court of last resort.

My treatments have been applied on all proximal surfaces. However, my most intensive work has been on the six upper anterior teeth, for the following reasons:

The solution is most easily applied here.

Fig. 13.—Treatment of proximal caries. When proximal caries is present, it is not always possible to place a filling at the time of discovery. A hole is drilled with a round bur distally to the marginal ridge. A part of the carious tissue is removed with the bur, the cavity is dried, the solution is applied and reduced, and the cavity is filled with cement. The cavity will be protected for weeks or months. In one case, protection was afforded for three years and the proximal surface of the next tooth remained immune, in a highly susceptible mouth. The opening shown is larger than would ordinarily be necessary.

It is most valuable here for the simple reason that we have no satisfactory filling material for these teeth. From an esthetic standpoint, these teeth constitute one of the prominent, beautiful features of the face. Results are more readily determined here.

A very limited knowledge of the sciences required for research; an insufficiency of time, so necessary to solve such a problem; a lack of opportunity to devote the necessary attention to the work—these and many other obstacles have prevented me from presenting a more conclusive proof. However, I feel justified in making the following statements:

This is the most effective method yet suggested for the control of proximal caries. I have yet to observe a failure where the treatment was applied while the continuity of surface remained.

If the solution did nothing more than disclose the presence and extent of the invisible caries, its universal use would be justified. Its use is feasible because of ease and simplicity of application. It is economical because of low material cost and the little time required. There is no danger associated with its use. To be sure, it must be kept off the lips and face, and the common fear of objectionable stains is to be kept constantly in mind. Research workers have told us how teeth vary in their capacity to absorb stains. In very rare cases, solid enamel will stain. In treating thousands of cases, I have found but three such cases, and in these the stains were easily removed.

INSTRUCTIONS FOR APPLYING HOWE'S SOLUTION

Proximal Surfaces of the Front Teeth.—I seldom use the rubber dam for children under ten years of age. If it is used for children and young adults, the teeth should not be ligated, for the ligature will do irreparable injury. Whether the dam is used or not, the teeth should first be cleaned and sprayed, equal parts of hydrogen peroxide and water being used. All débris and gummy material should be removed. If this cannot be accomplished with the spray, the débris should be removed with floss or very fine grit strips.

If the dam is not used, a napkin is placed over the upper lip, also one over the lower lip, both being held away from the teeth to be treated. (Fig. 1.) The teeth are dried thoroughly by blowing cold air

through each proximal space. The surfaces must be clean and dry. Next, a piece of ordinary waxed silk floss about 10 inches long is doubled in the middle and the loop put into the solution. The floss is now forced past the contact and the wet part brought against the proximal surfaces, being rubbed back and forth just gingivally to the contact point. It must be "rubbed" in as the painter brushes his paint into the wood fibers. (Fig. 1.) If there are any interstitial spaces caused by acid, they must be filled with the solution by friction. The same piece of silk may be used by re-dipping the same wet place in the solution and repeating the application at each space. If the silk frays, we may be sure there is a break in surface continuity and much damage has occurred. The results of the treatment, therefore, will be less sure. The surface is dried thoroughly as soon as the operator has finished rubbing the solution in with the silk. Cold air is used, as hot air may scare or burn the patient.

This is only one way of applying the solution. A wire loop, made for the purpose, or even a toothpick may be used provided it is "frictioned" in with floss.

If the best results are desired, the solution should be reduced with eugenol. The entire labial surface is polished with rubber cups, with a very fine abrasive. Ordinary pumice is too coarse. Triple X silex moistened with hydrogen peroxide makes a good polishing agent. (Fig. 2.) The stain is easily removed if it is polished immediately after the application. No fear need be entertained, as the only stain remaining will be on the etchings, where it should be.

Bicuspids and Molars.—For the posterior teeth, cotton rolls are used if the patient is too young to use the rubber dam. Air is blown between the teeth and the solution may be carried between the beaks of stainless steel cotton pliers, dropped on the contact point and carried on to the proximal surfaces with the silk floss as described for the anterior teeth, except that it is not necessary to dip the floss in the solution.

Usually, the bicuspids and molars may be treated while fillings are being made under the rubber dam. A number of teeth should be included in the dam so they will dry while fillings are being placed. The solution is applied and reduced, and is allowed a few minutes to penetrate before the dam is removed.

When the dam is in place and we are treating the bicuspids and molars, it is wise to flood the interproximal spaces. There is no danger in flooding these spaces and results will be better. I find it better to treat one side, or one-fourth of the teeth at one sitting. However, this is optional. We always consider it very neglectful if the dam is removed and every proximal surface which was included under the dam is not treated.

I do not know how often these treatments should be repeated. I do know that they cannot be repeated too often. Anterior teeth, for reasons stated above, should be treated more often.

While this work may be considered as being in its experimental stage, enough has been proved to warrant the immediate attention of the Research Commission.

There is great need for study on this subject. To those men who wish to join in such a valuable study, I make the following suggestions:

The decidious teeth should, of course be treated; but because they are in the mouth so short a time, it is better to begin on the first permanent molar.

The pits and fissures of the first molars are, of course, filled, for that is the one and only method for treating them.

The mesial surfaces of the first molars are treated to prevent proximal decay where these teeth make contact with the distal surfaces of the second deciduous molars.

As the incisors erupt and come into contact, they are likewise treated.

All pits and fissures in the bicuspids are filled soon after eruption.

All proximal surfaces are treated as soon as they erupt sufficiently to form proximal contacts.

The treatments are repeated uniformly every three months, or four times a year. Four or five years of study would, in my opinion, demonstrate that proximal caries can, to a large degree, be controlled.

DEEP-SEATED CARIES

For years, in our efforts to remove "every trace of decay," we have destroyed innumerable pulps. How could we assume that every trace was removed? How do we detect such tissue? We have stopped when we removed discolored or softened tissue. If it was not softened or discolored, we had no means by which we could know it was carious. If it was hard and not discolored, we concluded that it was sound structure.

It is necessary to use Howe's solution on only a few cases to learn how poor our methods are. Indeed, in some cases, a wall may have caries as much as a millimeter thick on it, and we could not, with our old methods, know that it was there.

This method has been a revelation to me. After I had used it for only a short time I began to see what I had been covering with my fillings, and I also began to see why my patients had recurrent caries, uncomfortable teeth and, later, dying pulps.

Since we began to fill teeth, we have overlooked the very important fact that acid is the active agent in the carious cavity. We cannot see the acid; we can see only the results of its activity. How, then, I ask you, may we hope to remove the acid with a bur? Not only have we been failing in removing carious tissue, but, what is worse, we also have failed to reckon with that acid of caries which precedes the whole process. It is quite apparent, therefore, that to stop a carious process, we must destroy the acid organisms, the acid makers, and neutralize their acid.

When Percy R. Howe added ammonia to a solution of silver nitrate, he alkalized it, giving it a pH of 8.5 to 9.5 (according to Samuel M. Gordon); which is, as you know, definitely alkaline. When this solution is applied to dried carious tissue, surface tension draws it into the interstitial spaces, where there were once mineral salts, which have now been dissolved out by the acid of caries.

When eugenol is added, the solution is changed to silver albuminate, silver oxide and, possibly, a colloidal silver. What is perhaps most important of all, the acid of caries is neutralized. When this is understood, those men who, like myself, have been using the Howe solution since it was introduced, can understand why an acid-soaked cavity may be changed from an inoperable condition to an operable one, and how a pulp irritated from the acid of caries can return to normal. When the treatments are repeated in these deep cavities and temporary cement is inserted, the pulp is given time to lay down secondary dentin for its protection against the oncoming cavity of decay. (Figs. 3 and 10.) Those operators who cut into these deep, sensitive cavities with no preliminary treating have results ranging all the way from a slight discomfort to a final pulpitis and pulp death.

When the dentist learns the use of this solution, he develops a new concept and enjoys a new experience in operative work. He will have less use for procaine. A slight pulpitis in a child's molar will give him no anxiety. He will learn that he no longer needs to expose pulp horns and cap pulps in an effort to "remove the last trace of decay," when he understands how he may transform the decalcified organic matrix of carious tissue into a hard, impervious, metallized, alkalized, insoluble tissue which can be burnished like amalgam. (Fig. 4.)

This is accomplished by repeated treatments. In the severe cases, especially in pulpitis, the first and second treatments may consist simply of flushing the cavity with warm water; drying by packing

cotton in the cavity for a minute, and removing this and placing in the cavity a pellet of cotton saturated with the solution, over which is placed a larger pellet saturated with eugenol. The patient is dismissed for twenty-four hours. Later, the treatments are continued under the rubber dam, with a week or more between treatments. Each time, some of the carious tissue is removed, preferably with a large, round sharp bur; after which the solution is again applied, dried with warm air and reduced with eugenol. If the decay is deep and there has been some pulpitis, treatments are more frequent and more time between the treatments is allowed.

PITS AND FISSURES

Pits and fissures cannot be immunized with this solution. I tried for five years to immunize them and failed. Decay begins here at the dentino-enamel junction and the solution will not reach that point. However, it is always wise to apply it as a disclosing solution only. In this way, it is possible to see the faults and include them in the cavity outline when the tooth is filled.

FORMULA

Howe's Ammoniacal Silver Nitrate Solution is made as follows:

To three grams of clean silver nitrate crystals, add 1 cc. distilled water; heat and stir with a glass rod until dissolved. Do not boil.

When the crystals are all dissolved, the solution is allowed to cool to room temperature; then 2.5 cc. of strong 28 per cent ammonia is slowly added. This will throw down a black precipitate which is soluble in ammonia. This is not wholly dissolved, for, if this occurs, an excess of ammonia will result. The solution is then filtered.

The solution is very sensitive, and a small amount of contamination or variation in the chemicals may change the result. For a reducing agent, where one is required, use eugenol.

Dr. J. W. Va Verka, Professor of Chemistry, Creighton University, Omaha, Nebraska, says:

If formalin is used as a reducing agent the basicity of the original solution is overcome and tends to leave the system with a pH below seven, well on the *acid* side, due to formic acid formation. Another thing to be noticed, is the fact that the silver is not precipitated in the metallic state to a large degree, but rather large amounts of silver oxide are found to be present.

When eugenol is used as a reducing agent a slower reduction takes place, but at no time was the pH found to be on the acid side—rather a pH higher than seven resulted (alkaline). The reduction of the silver is more nearly complete, with a fine, shiny, mirror-like surface being produced.

He also offers the following precautions:

1. Avoid touching the crystals with the fingers or other organic materials.
2. Use only transparent, uncolored crystals.
3. Accuracy in weighing.
4. Use freshly distilled water.
5. Use clean glass receptacles for dissolving and check for precipitates by holding sheet of paper behind the transparent vessel.
6. Store in clean amber bottles with glass stoppers. Avoid light.

A fresh supply can be obtained from the druggist every ten days. A small amount is all that is required to last ten days. A small amount of the silver nitrate solution and of the eugenol should be put in dappen dishes for each treatment, so as not to contaminate the stock bottles.

Stains on fingers, nails, linen and face can be removed with iodine or saturated solution of potassium iodide.

CONCLUSION

Caries results from the presence of acid-forming microscopic bacteria. Neither the bacteria nor the results of their activity are visible during the onset, which covers an indefinite period of time. On proximal surfaces the time may be as short as one or two years in the mouth of a child, but may be many years in the mouth of an adult. All this time, it is an unseen enemy.

The attack on caries must be made with the full knowledge that it may be there, but unseen. (Figs. 6, 7 and 8.)

The use of Howe's solution brings the carious area definitely within our vision, provided the surface is not hidden. It is during this unseen stage and before there is loss of surface continuity that this treatment is effective in caries control. It is feasible because this period covers a considerable period of time. If it is applied before the surface is etched, it seems to prevent the etching.

If this treatment is only half as effective as its proponents believe it to be; if it will check but 10 per cent of the etchings; if it does nothing more than definitely disclose invisible, incipient carious spots, it merits universal use.

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THE SURGICAL PREPARATION OF THE MOUTH FOR FULL DENTURE RECEPTION

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ANY operation, whether it be in the field of medicine or in that of dentistry, should be designed to benefit the patient and should be performed only in the presence of conditions that require correction. The object of any surgical procedure within the mouth should be the elimination of abnormal or pathologic conditions. Because surgery of this type frequently involves the loss of teeth, with consequent impaired function, careful consideration must be given to the physical, mechanical and esthetic results. The construction of artificial dentures on a base as near the normal as possible necessarily becomes an important part of such a procedure.

COOPERATION BETWEEN ORAL SURGEON AND PROSTHODONTIST

Many of the unsatisfactory results of surgical preparation of the mouth for dentures have been due to lack of understanding of the demands and limitations of prosthetic service by the one who has removed the teeth. Likewise, the dentist who is to restore the mouth has failed to recognize the surgical problem, and has refrained from referring the patient for such service in fear that a radical operation will be performed. Cooperation between the oral surgeon and the prosthodontist is therefore imperative if the best result and an increased benefit to the patient is the objective.

It is a fact recognized by leaders in the prosthetic field that, in the removal of all teeth, an operative technic should be employed which will facilitate the early insertion of dentures upon a base free from the rough, jagged edges of bone that are too often left for Nature to smooth after simple extraction. The desire for this result is to be found in the dental literature of many years ago, when extracting forceps were used to bite off sharp bony projections. As the history of the development of this service into an accepted dental procedure has been published elsewhere, no effort will be made here to review this literature or to give credit to those who have contributed so much to this type of surgery.

The removal of any tooth is a surgical procedure, requiring the same surgical principles as are followed elsewhere in the body. It is the operator's obligation to remove all teeth with the least amount of trauma possible, to eradicate pathologic areas resulting from removal of teeth and to remember that he is preparing the foundation upon which the patient will necessarily wear a denture. It is imperative that the operator follow a definite technic in order that the patient shall receive maximum benefit and be assured the same result as might be expected of surgical measures in any other part of the body.

It is impossible to lay down iron-clad rules to be followed in every case, but the importance of a predetermined plan to be followed as a basis of operation must be emphasized. A correct diagnosis is essential to all successful treatment and must be made before any operation is attempted.

When a patient presents himself for the

(Read before the Section on Full Dentures at the Seventy-Third Annual Midwinter Clinic of the Chicago Dental Society, February 16, 1937.)

removal of all remaining teeth, the diagnosis involves a consideration of the elimination of all pathologic conditions which may affect the future health of the patient. Here, it is necessary to depend on roentgenographic findings, not only of the teeth that are present, but of all edentulous areas as well, and their co-relation with clinical signs and symptoms, together with a careful history of the patient's general physical condition. The diagnosis cannot be complete without consideration of the mechanical and esthetic results, which can be determined only by one who is thoroughly familiar with the prosthetic demands and limitations of the case. This, of course, involves the future health and well-being of the patient and is the responsibility of the one making the restoration. A lack of cooperation or failure to understand the various problems involved have been responsible for many of the difficulties that have been encountered.

The obtaining of preliminary photographs, records of the dimensions of the face, length, color and alinement of the natural teeth and the preparation and articulation of casts for future reference, as advocated by many leaders in the prosthetic field, constitutes an important part of this preliminary study and is of great value to both the surgical and the restorative service.

PREPARATION OF MOUTH FOR FULL DENTURES

The amount of surgical interference necessary in the preparation of the mouth for full denture reception may be determined by:

1. Pathologic conditions, such as are revealed by a complete set of roentgenograms, clinical examination and history.

2. Esthetic requirements. The reduction of the labial and buccal alveolar bone should permit the proper alinement of the artificial teeth so as to restore normal facial contour and prevent undue fullness of the lips or exposure of an excessive amount of denture material with changing facial expression.

3. Mechanical requirements, such as the reduction of large bony protuberances, excessively heavy alveolar crests or large bulky tuberosities, which may interfere with securing an accurate impression or the proper seating of the denture.

4. Ridge relation, or the relation of the upper and lower ridges to each other. If the upper and lower ridges are not in proper relationship, surgical correction will assist in harmonizing the ridges and permit a more nearly ideal articulation of the artificial teeth. Abnormal opening of the bite with artificial dentures may also be prevented by proper mouth preparation.

5. Abnormal muscle or frenum attachments. Surgery of the soft tissues is as often necessary as the reduction of the osseous structures. Frequently, muscle and frenum attachments must be modified in order to permit better retention of the denture as well as to insure normal facial expression.

6. Ridge condition. The presence of soft flabby ridges, resulting from excessive hypertrophied submucous tissue or atrophic changes in the alveolar ridge, will, if not corrected surgically, seriously affect the stability and efficiency of the full denture.

All these factors being duly considered, the necessary operation is performed in accordance with the requirements of the individual case. In certain patients, the teeth may be removed before incision is made or the soft tissues are reflected from the labial or buccal surfaces. In other cases, it is advantageous to make an incision about the necks of the teeth and over the crest of the ridge distally to the last tooth, sufficiently far to permit the reflection of a mucoperiosteal flap. Such a procedure affords a clear view of the osseous structures and protects the soft tissues from injury during operation.

In cases wherein difficult extraction is expected owing to the presence of a heavy dense alveolar process or excementosed or

curved roots, the removal of a portion of the labial or buccal alveolar bone surrounding the teeth is indicated. This facilitates the removal of the teeth and prevents fracture of large portions of buccal or labial bone from the force necessary for extraction. Where destruction of bone has been excessive, as in cases of extensive pyorrhea, the removal of the teeth and pathologic tissues, followed by the smoothing of sharp jagged edges of bone, is all that is necessary.

After removal of the teeth, the alveolar process in the anterior portion is reduced by arching the labiolingual contour from a point just above the high lip line to the palatal or lingual edge, which is also trimmed to approximately 1 to 1.5 mm. below the lingual gum margin. This form of preparation will prevent occurrence of the high thin ridge which has been the subject of so much criticism following attempts to reproduce the so-called V or U type ridge.

In the posterior areas, reduction of rough jagged edges of bone, large bony protuberances and bulky tuberosities is indicated. The contour of the posterior ridge is prepared by an arching from buccal to lingual sides, similar to that of the anterior portion. Reduction of these areas should be made from a point midway between the highest point of reflection of the labial and buccal pockets and a line corresponding to that of the high lip line. Such a preparation will result in a rounded arched ridge which will present the greatest surface at right angles to the long axis of the artificial teeth.

This procedure was advocated some time ago by J. W. Crawford, of Milwaukee,[1] who also states that it results in a better ridge to receive the stress of mastication and more closely conforms to a shape between the V and the U type ridge, with the labial or buccal surface forming one arm of the figure and the palatal or lingual the other. Dr. Crawford has also stated that because the force of mastication is on a line with the long axis of the tooth, the edentulous surface with the greatest area at right angles to the lines of force affords the greatest stability for the full denture.

Frequently, there is a large amount of thick fibrous tissue overlying the tuberosities, which, if not removed, interferes with the articulation of the artificial teeth. Such abnormal tuberosities should be reduced by excising a V or wedge-shaped section of tissue, compressing the buccal and lingual edges and retaining with a suture.

Trimming of the gum tissue is an important step in the surgical preparation of the mouth for dentures. This is particularly true when there has been rather extensive reduction of the osseous structures in the anterior region and is necessary to prevent folding or puckering in the replacement of the soft tissues. If it is not done, the result may be a loose flabby ridge which seriously interferes with the stability of the denture. In the posterior areas, removal of unhealthy gum septa and infected pockets that remain attached to the mucoperiosteum is always indicated.

The thick fibrous gum tissue surrounding the teeth should be preserved as long as it is healthy. Such tissue forms a better denture base and protects the ridge from the stresses of mastication following construction of the artificial restoration.

"Surgery of the Soft Tissues of the Mouth," was very ably presented before the oral surgery section at the last midwinter meeting of the Chicago Dental Society by D. E. Woodard,[2] of Kansas City. Dr. Woodard described procedures designed to obtain better results in shape, contour and stability of the edentulous alveolar ridge. He discussed also the correction of prominent areas following removal of isolated teeth, and illustrated a technic for replacement of soft tissues in conjunction with the preparation of the mouth for dentures. His presentation included a procedure to be followed for the correction of flabby ridges or low muscle at-

tachments, for patients who are unable to wear dentures satisfactorily.

Frequently, the labial frenum extends over the ridge or its attachment is so low that it will interfere with the proper retention of the denture. Surgical removal of the frenum in such cases greatly assists the patient in wearing the denture.

The amount of suturing necessary depends on the replacement of the soft tissues to obtain a properly prepared ridge. Some cases require more sutures than others, particularly those in which a mucoperiosteal flap has been reflected. Great care must be taken in the placing of sutures in order to prevent a change in the mucobuccal fold from attempting to approximate the buccal lingual tissues. It is always well to determine the amount of tension to be placed on the sutures by holding the tissues in the labial and buccal folds in proper position and examining frequently to be sure that there is no displacement.

Occasionally, mouth preparation has been criticized as having resulted in the removal of an excessive amount of alveolar bone, when, as a matter of fact, the mucobuccal fold has been changed by improper suturing. The use of splints in the form of immediate dentures, or otherwise, sometimes serves to avoid abnormal change in these tissues. All sutures should be removed early. This will avoid unnecessary irritation of the soft tissues, as sutures are usually necessary to retain these tissues in position only during the first thirty-six to forty-eight hours following operation, during which time a blood clot is formed.

POSTOPERATIVE CARE

After removal of the sutures, the use of a mouthwash such as hydrogen peroxide and warm water, equal parts, two or three times daily, greatly assists in the recovery of the superficial tissues. This should not be used immediately after the operation. Such an oxygen-liberating mouthwash prevents the propagation of anaerobic organisms, which interfere with normal healing of mouth wounds.

The construction of an artificial denture before the removal of all teeth, with insertion immediately following extraction, affords excellent results in many cases. Such prostheses are usually referred to as immediate dentures. The advantages and comfort to the patient from such a service have been presented rather extensively by various writers in the prosthetic field. The procedure to be followed is based largely upon pathologic conditions present and the age and the general physical condition of the patient, as these conditions govern the number of teeth that can be safely removed at one operation and the time that necessarily must elapse before the restoration can be completed.

SUMMARY

I wish to emphasize the desirability of:

1. Closer cooperation between the oral surgeon and the prosthodontist.

2. Complete oral diagnosis pertaining to physical, mechanical and esthetic results.

3. A predetermined plan of operative procedure for both surgical and restorative service.

4. The recognition of conditions requiring surgical interference: (a) pathologic, (b) esthetic, (c) mechanical, (d) ridge relation, (e) abnormal muscle or frenum attachments and (f) the presence of soft flabby ridges.

5. An operative technic as a basis of operation to insure a properly prepared mouth with well-rounded arched ridges which will present as much surface at right angles to the long axis of the tooth as it is possible to obtain.

6. Immediate dentures in any case where the patient's age and physical condition will permit, and where there can be close cooperation between the oral surgeon and the prosthodontist.

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℞ DENTIFRICE

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WHEN the question is asked by the patient, "What is a good dentifrice?" how many of us have before us the indications for the specific dentifrice and prescribe accordingly? Many dentists will reply that "most of the dentifrices are good, but the brush must be used in the proper manner, following this statement with some instruction on proper brushing. Others will merely name one or two of the popular brands of dentifrices.

The majority of dentists fail to grasp a golden opportunity that comes with the question, "What is a good dentifrice?" At that moment, the patient is wholeheartedly seeking advice. He may have, at some time during the operative sessions, attempted to tell us how to do this or that, tactfully dropped innuendoes concerning various technics. He may have openly complained about certain procedures and objected to our recommendations; but now he asks our advice. What does he think when his dentist, with a shrug of the shoulders, merely offers an opinion which could have been given by the person sitting beside him in the train or bus, or by the boy at the corner stand?

When our advice is sought on a dentifrice, why not be prepared, and, after noting certain peculiarities of the patient's teeth, soft tissues and saliva, prescribe the dentifrice that is indicated?

As yet, there has not been a "cure-all" dentifrice made and sold under a trade name. Many of our patients, however, believe that such and such a powder or paste is just the thing for all ailments of the teeth. Our medical confrères soon are cognizant of the fact, after examination and mental listing of the symptoms, that the "cough medicine" made by such and such a pharmaceutic house is not the remedy for the cold that has sent the patient in for medical treatment. That proprietary product cannot act in the same way for the severe head cold as it would for a deep congestion of the lung. After taking into consideration all the symptoms, both subjective and objective, the physician is ready to prescribe carefully prepared and accurately weighed combinations of drugs which are indicated in the treatment of the physical ailment that he has diagnosed.

Let the dentist follow the same procedure. He has noted in the oral cavity certain symptoms of a lack of hygiene, or the failure of the dentifrice being used to promote oral cleanliness. The source of the present lack of oral hygiene may be easily detected by examination.

The operative and prophylactic work having been completed, we are now ready to advise the patient what dentifrice to use in order to keep the teeth and surrounding soft tissues clean. We find that the patient has a thick saliva with an excess of mucin. On inquiring of the patient as to his success in cleaning the teeth, we are told that a film or coating forms on

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the teeth a short time after the brush has been used.

Harold K. Box[1] and Hermann Prinz[2] advocate for such a condition the use of a dentifrice which, when used in solution, will assist in the removal of most of the mucin from the mouth. It should be borne in mind that mucin plays a definite part in the health picture of the mouth and teeth. The use of strong agents, such as acids, which will cause the complete removal of mucin from the mouth, is not desirable.

The proportions of such agents as the commercial product guarantees to remove mucin or film should be determined, not by the mixing department of a toothpaste factory, which makes its product by the ton, but by the dentist.

The dentifrice advocated by Box is as follows:

℞

Phenolphthalein 2 grains
Saccharin [gluside] 7 grains
Menthol 15 grains
Calcium oxide 100 grains
Sodium chloride 1 pound

Sig. One-quarter teaspoonful to one-half glass of warm water.

Stillman's method of brushing is used, the brush being dipped into the solution from time to time.

The formula for the same type of dentifrice as recommended by Prinz is as follows:

℞

Sodium chloride 1 pound
Calcium oxide 150 grains
Menthol 15 grains
Saccharin [gluside] 12 grains

Sig. One-third teaspoonful in a glass of warm water.

This may be used as a dentifrice by repeatedly dipping the brush while cleansing the teeth. It may also be used as a mouthwash.

In the routine prophylactic care of the mouth at the chair, we may find that the cemento-enamel at the junction is very thin, and from either subjective or objective symptoms, we learn that there are many areas of extreme sensitiveness and advanced erosion at the gingiva. Bodecker[3] calls this a form of decalcification, and treats the condition with a remineralization powder suggested by Andresen.

The formula for this dentifrice is as follows:

℞

Tartaric acid 448 grains
Solution of gelatine 10% 50 gm.
Mix and add
Precipitated calcium phosphate
Precipitated calcium carbonate
Basic carbonate of magnesium āā 160 gm.
Mix dry and add
Sodium bicarbonate 560 gm.
Sodium chloride 160 gm.
Corrigents of taste, ad libitum
Mix dry.

Eight ounces of the powder will last one person three months. Bodecker states, "The purpose that this powder is to fulfill is to go into solution in the saliva and in this state permeate and recalcify the porous areas in the enamel."

Bodecker's article should be read carefully and with an open mind as regards the use of the remineralization powder in children. To say the least, clinical experimentation should suggest itself to the reader as to the ability of this dentifrice to check early decay in deep pits and fissures.

The patient in the chair at the moment may be a new one in the office and he may inform us that it has been his practice to visit a dentist every three or six months; or he may be a regular visitor to our office at three- to six-month intervals. On close examination, we find the unmistakable signs of an extremely acid saliva. The simple litmus paper test or one of the more detailed methods may confirm the diagnosis. In a case of this type, the use of a strongly alkaline dentifrice is indicated.

Stillman and McCall[4] recommend the following "alkaline tooth powder":

℞

Calcium carbonate 4 ounces

Castile soap 4 drams
Calcium hydroxide 4 grains
Sodium phosphate (dibasic) 5 grains
Saccharin [gluside] 6 grains
Flavoring oils q.s.

In the event there is a heavy deposit of salivary calculus, not only near the sublingual and submaxillary ducts, but also on all surfaces of the teeth, careful investigation should be made in order to determine the etiologic factor or factors.

The condition may be caused by an excess of mucin in the saliva plus an excess of calcium, this combination coming in contact with fermenting bits of food. The condition may be caused by mouth-breathing, the chilled air causing the calcium to leave the solution; or a remote condition, such as a blood dyscrasia or diabetes, may be the etiologic factor in the heavy deposit. Again, the condition may be caused by a sublingual secretion heavily laden with calcium coming in contact with an already acid residual saliva.

Locally, the presence of heavy accretions is plainly manifest, and we are obliged to give much thought as to what dentifrice will control or remedy the condition. The cooperation of the physician is imperative at this point, if oral hygiene is to be maintained.

It is then within our scope of treatment to experiment with either a mucin-counteracting dentifrice (the formula of Box or Prinz) or one which has a strong alkaline reaction (the formula of Stillman and McCall). When the patient has been advised that we are attempting to establish greater oral cleanliness, his cooperation is most willing during the few weeks of experiment with the two types of dentifrices.

CONCLUSION

Let me again emphasize the fact that our patient's demands must be met if we, not the radio announcer or the advertising companies, are to be known as the teachers of dentistry.

When the dentist prescribes a dentifrice for his patient, he makes dentistry more of a real healing profession and further from an organization of strict technologists. Stillman and McCall[5] state that "Dentistry has for many years concerned itself with technological procedures, and by so doing, it has earned for itself the name of being a mechanical profession." Of course, the technologic phase of the profession must remain and improve, as it no doubt will; but let us not lose sight of the ability we all possess, acquired through undergraduate work and postgraduate study, in the field of prevention. THE JOURNAL, in an editorial,[6] quotes from the 1936 report of the Surgeon General of the United States Army: "Dental service cannot function as a true health service until progress is made in the prevention of dental diseases." Prescribing for our patients to eradicate one universally accepted cause of dental diseases, namely filth, will do much to assure a more favorable report in future years.

Preparing ourselves to be ever ready to serve in the manner herein recommended brings home the satisfying thought that each time the patient selects a dentifrice, his dentist, and not the actors in the radio sketches or the artists who paint the billboards or magazine beauties, is playing the important part in the fight against decay of the teeth.

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SPEECH DEFECTS AND RELATED ORAL ANOMALIES

By James Sonnett Greene,* M.D., New York City

ONE never realizes how far-reaching a mouth condition can be in its influence on personality. To illustrate, let me read an abstract of one of the many letters received at the National Hospital for Speech Disorders:

At 21, I am completely and thoroughly broken and there is nothing in my life but speech trouble and shame. I live like a hermit. I have reached the end of my rope and unless you help me, I can see no way out but suicide.

I was given psychiatric treatment at B. Hospital, and at the beginning of this month I asked for and have been receiving hypnotic treatment three times a week. In spite of all this and many other things I have tried, I am still suffering.

My speech to me is the most important thing in the world. Please help me!

Would any one imagine that there could be any connection between all this condition and a broken tooth? Yet there is.

Following is the case history of the patient who wrote this letter:

H.G., aged twenty-one, came to the clinic with a speech defect and presenting a personality problem. He was slovenly and careless about his personal appearance. The first thing that impressed us was the fact that he tried to speak with his mouth closed and that he never smiled. Examination revealed, among other things, that he had a broken incisor which caused a certain amount of physical disfigurement. This broken tooth later proved to be a very important contributing factor in his personality involvement.

As a young child, he had fallen and sustained this mouth injury. As time went on, he became so sensitive about his disfigurement that, in social situations, he tried to hide it by keeping his mouth tightly closed. Eventually, this habit resulted in an inhibition which finally became the most important factor in his problem. This same apparently insignificant defect served as a most important determinant toward a life dominated by introversion, evasion and marked inadequacy. Ultimately, his personality became so warped that he had no special contacts with the world about him. Consequently, there was present a composite dental, speech and personality problem. The solution was obvious. A composite dental, reeducational and social therapy was instituted. The patient's personality underwent a complete change. He now smiles and speaks well.

SPEECH

Speech is articulated voice. Voice itself has been defined as a column of breath set in vibration by its own impact with the vocal cords and reinforced by diffusion through the various resonators into the surrounding atmosphere. When voice is molded into syllables and words, it becomes speech. Thus, we see that speech is produced by a combination of two distinct mechanisms, the vocalizing and the articulatory. The first mechanism includes the vocal cords and all the respiratory organs; the articulatory mechanism includes the upper portion of the larynx, the pharynx, the palate, the tongue, the teeth and the lips.

Voice is the medium for the expression of the emotions. Intelligence converts it

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into speech by bringing about changes in the various parts of the vocal tract. When an anomaly of some part of the articulatory mechanism makes it impossible to bring about normal changes in the vocal tract, the result is speech of an abnormal or defective nature.

The tongue, on account of the prominent part which it plays in articulation, has always been considered the most valuable and important instrument in the production of normal speech. Nevertheless, it has been demonstrated many times that defects of the tongue very often produce only minor defects of speech. Even with the total loss of the tongue, it has been shown that one can speak fairly well, though some of the sounds will be missing.

This has been accomplished by the substitution of the other parts of the mouth for tongue action. Thus, for example, in the correct formation of the sounds *k* and *g*, the back part of the tongue comes in contact with the back part of the palate; but the sounds *k* and *g* may be simulated by producing, instead of this tongue action, a glottal catch far down in the throat, above the exit of the larynx. The consonants *t*, *d*, *n* and *l*, which depend mainly on the contact of the tip of the tongue with the hard palate, can also be produced when a tongue defect exists by substituting lip action for tongue action. If one retracts the lower lip and presses it against the alveolar margin of the upper jaw, the glottal catch is made just as if the tongue were used. The sound of *l* is heard fairly well even when there is a complete loss of the tongue, since remainders of the genioglossus are always able to lift the bottom of the mouth, thus forming an impediment in the center of the emanating air column.

The *s* sounds, although not formed in so exact and sharp a manner as when the tongue is used, can be made sufficiently clear by the correct approximation of the upper and lower teeth. The sound of *r* very often causes difficulties, but a labial or laryngeal *r* can be readily substituted for the lingual sound. The labials, *b*, *p*, *m*, *f*, *v*, *w*, can be produced naturally because the tongue is not used at all in the formation of these sounds.

From these examples, one can readily deduce that even if the tongue is completely extirpated, the power of speech is not necessarily lost. The tongue is not the essential factor in articulation. However, the tongue is an important factor both in articulation and in determining the form of the lower jaw.

In reference to the lips, the outposts of articulation, there is little to be said. Defects of the lips cause disturbances of speech only when they seriously affect the articulatory mechanism.

RELATION OF THE TEETH TO SPEECH

Likewise, the existence or non-existence of teeth is not so important for good speech as the shape and roof of the mouth, which acts as the sounding board, and on which normal speech greatly depends. Almost all speech sounds, except the hissing sounds of *s* and *z*, can be produced after a fashion without the use of the teeth. But the relation or connection between the palate and teeth is so close that therapy of the palate without therapy of the teeth, or vice versa, cannot be considered.

Now let us see why there is such an intimate relationship between the teeth and the palate.

We distinguish anomalies of the shape, number and position, as well as structure, of the teeth. Most important to us are the anomalies of position, because they interfere with the normal biting ability and because they bring about changes in the palate.

One of the most frequent of these anomalies is protrusion of the lower jaw, otherwise called underbite or mesial occlusion. Usually, the lower jaw is too large or it is pressed a little forward out of its regular articulatory position. Quite

often, this is caused by habit or by imitating the speech of others. In a very pronounced underbite, *f* is often formed between the lower row of teeth and the upper lip. (Such an *f* formation is seen in children having a double harelip, in which the upper jaw is generally retarded in growth and the lower jaw protrudes. The retraction of the upper jaw depends on the tight scar of the double harelip.) Also, the underbite can be so pronounced that the articulation between the lower row of teeth and the upper lip is even applied to the labials *b, p* and *m*. When this is the case, the lower lip protrudes so far that it seems to be hanging free in the air, giving the face, especially in profile, a very awkward appearance.

Another anomaly of tooth position is the projection of the upper row of teeth, so-called overbite or distal occlusion. Variations occur at the overbite, technically designated as Class 2, as well as at the underbite, or Class 3. When there is a prominent overbite, as when the incisor teeth of the upper jaw protrude very much and the upper teeth rest upon the lower lip almost constantly, the patient looks as if he were always pronouncing the letter *f*. It is naturally very difficult for him to coaptate the lips, and consequently it is almost impossible for him to make the labial sounds *b, p* and *m*. Furthermore, the upper lip, no longer used for closing, leaves the upper row of teeth uncovered and always visible to the eye; which makes one look very unattractive.

Still another abnormality is the open bite, which is due to the fact that the alveolar process of the molars is too large. The front open bite is well known to all of us. Less consideration has been given to the side open bite, which is frequently found in certain speech disturbances.

Besides these anomalies of tooth position, a dislocation of a faulty tooth may also hinder the coordination necessary for speech. For instance, if a front tooth is pressed backward out of alinement, the laying of the tongue on the alveolar margin may be hindered and a hissing sound produced where there should be a closed or stopped sound.

In this connection, it is well to realize that speech plays an important rôle in producing a normal condition of the alveolar process and normal position of the teeth; for before eruption of the permanent teeth, the teeth have but a shifting position in the alveoli. The regulation of the teeth is aided through speech by the concerted action of the lips and tongue, the lips pressing on the teeth from one side and the tongue from the other. This is contrary to the general idea that the teeth have an influence on speech instead of speech having an influence on the teeth. The process is similar to that which occurs in the formation of the head, the form of the skull depending on the growth of the brain and *not* the growth of the brain on the form of the skull.

The most frequent cause of the anomalies of the positions and cross positions of the teeth is a lack of space. The alveolar process is too small for the teeth, but orthodontia can readily take care of that. This lack of space may have the following effects: It can cause a pushing apart of single teeth from the row, and it can produce a curving of the alveolar arch horizontally or vertically. The pushing of single teeth from the row may produce defects of articulation which are of minor importance, but the arch positions are of great importance.

Observing the teeth of the upper jaw from below, one notes that they are placed in a more or less oval half circle; in other words, in a horizontal arch. If, for some reason, the teeth of the upper arch do not properly articulate with those of the lower arch, lateral gaps are formed. These gaps prevent the tongue from correct approach and cause faulty noises in speaking.

The relations are more obvious with the vertical curves. The vertical curve, as we observe in the open bite from the

front, hinders the natural tight closing of the teeth necessary for enunciating *s* and *z*, causing a *th* sound in place of an *s* sound. Abnormalities of the teeth, loss of alinement, articulatory disturbances, all affect the production of the *s* sound.

SIGMATISM

The term sigmatism, chiefly used abroad, or the term lisping, more frequently used in this country, is applied to this inability to produce the friction sounds *s*, *sh* and *z*. Sigmatism is always attributable to a wrong position of the tongue, which, in turn, is occasioned, as pointed out, by anomalies of dentition and maxillary articulation. The defect occasionally originates in the period of second dentition. It develops preferably when the upper or the lower incisors are missing. Young children very often have this defect, if eruption of the incisors is delayed. This defect may be permanent, but if due to the second dentition, it is usually transitory, provided the eight incisors are completely formed.

There are various forms of sigmatism:

1. Interdental, wherein the patient, on account of an open dentition (cross-bite, interdental spaces, drifting of teeth, etc.), says "tshop" for "stop."

2. Addental, wherein the patient says "thoap" for "soap." A protruding lower jaw is a source of predisposition, as is also the front open bite.

3. Lateral sigmatism, due to a unilateral or bilateral inward or outward displacement of the jaw, causing an excess escape of air, the patient saying "sheven" for "seven."

We thus see that when teeth are out of alinement, they generally impede the motion and formations of the tongue, especially when there is a high palate. The sounds of the second and third articulation sphere are more or less strongly impeded, the *s* sound more so than the other sounds.

An interesting difficulty is often encountered when repair of the teeth causes great difficulty in speaking. The patient has to become used to the new proportions of space for his tongue until he again speaks as usual; but it sometimes happens that, in cases of this kind, the speech difficulties become permanent. For example, let us presume that the tongue is so near the alveolar margin of the upper jaw that, with the color experiment, only a very narrow groove appears, and that a bridge for the upper incisor teeth makes the space for the tongue still smaller. The air current cannot pass through the groove needed for the *s* formation, the tongue will lie too close to the foreign body and a lisp will be heard.

These cases are very annoying to the dentist because he is blamed for the condition. But blame is of course unjustified unless a full guarantee against speech disturbance has been given. In cases under contention, the remedy is simple enough. The patient is reeducated in the proper adjustment of the tongue, and the speech disturbance disappears.

Very often, if the teeth are out of alinement, unnecessary hissing noises are heard when *t* and *d* are spoken. I have had cases in which a side incisor tooth, pushed back out of the row, hindered the tight contact of the tongue with the alveolar margin of the upper jaw, an *s* sound being produced instead of the *t* or *d* sound.

Defects in enunciation can also be caused by the condition of the arches, especially the side arches. In 92 per cent of all cases of lateral lisp, the larger arch is on the lisping side. This would seem to indicate a certain relationship between the side arch and lateral sigmatism.

Now a word about the interrelationship of the position of the teeth, the roof of the mouth, the tone of the voice and the quality of speech. Without doubt, the roof of the mouth has a very great acoustic influence on the tone of the voice and the quality of speech. The general clearness of the vowels and consonants is affected by the form of the roof of the

mouth; and in the relation of the teeth to speech, the form of the hard palate must always be taken into consideration. The height of the palate depends on the development of the nasopharynx. Every case of high palate demonstrates a certain amount of respiratory incapacity and local infection, and this in turn always has an influence on voice and speech.

The question of voice and speech in cases of cleft-palate is a matter of vast importance. Before operation, the voice is seriously impaired by the malformation of the mouth and nasal resonators. In addition, the soft palate, which normally checks the air passing into the nasal cavities and comes into play through contact with the tongue in the production of the *k* and *g* sounds, is non-existent.

If the hard palate is cleft, consonants depending on the contact of the tongue and hard palate, such as *t, d, n, r* and *l*, are mutilated. No doubt you have noticed, when making various kinds of plates for patients having congenital clefts of the palate, that speech was more or less affected. But I wish to point out that the severity of the speech or voice defect does not coincide with the size of the palatal defect. There are small defects, small openings which one sometimes sees at the junction of the hard and soft palates, which interfere greatly with the production of speech. On the other hand, in some large defects of the palate, even with harelip on both sides, fairly good speech can be produced without resorting to mechanical interference. Usually, some existing condition of the nose or throat is responsible for this anomaly. An enlargement of the nose and its parts, or adenoid vegetation in the nasopharynx, may act as a substituting medium for the function of the missing palate, and thus make possible the formation of speech sounds.

Operations for repair now have good results. In discussing operative measures, Dunning[1] says:

1. DUNNING, H. S.: Question of Cleft Lip and Cleft Palate, radio address, May 28, 1936.

The cleft in the bone in front of the mouth should be closed in the first few weeks, if the baby is doing well. The opening or split in the lip should be closed two or three weeks after the bone operation, if the baby is in good condition. The palate should be repaired during the third or fourth year.

Delaying the operation until the child is 12 years old or so, generally makes it more difficult. And in some cases it has made it impossible to close the cleft in the palate, as it has become wider with age. In many cases, however, adult age does not interfere in obtaining good results, and we might say that it is never too late to operate on these cases if they are well and strong. Many have been operated on after 40 years of age.

There are many types of operations for the closure of the palate, but I would say the main object of all operations is to obtain for the child a long, soft, resilient palate that will close off the back of the nose and will do away with the typical nasal, cleft-palate speech.

The question whether the closure of the cleft remedies the defective articulation can be emphatically answered in the negative; but I might add that the resonance of the voice becomes more nearly normal simply through the improved anatomic status. In nearly every case, speech training is not only advisable but is also absolutely necessary.

After the operation, the cleft-palate patient usually presents one or all of the following defects of speech:

1. The familiar nasal resonance and nasal snort. These are due to the lack of elasticity and mobility of the soft palate. The levator muscles cannot raise the palate with sufficient power. The soft palate ordinarily would have regulated the exit of air through the anterior nares, but because of its inefficiency, the alae of the nose come into play and become compressed, more or less severe facial distortion resulting. This condition is the most difficult to overcome.

The production of the consonant *s* is nearly always accompanied by this unpleasant nasal sound or snort. After an operation, it occasionally happens that

the soft palate is too short to reach close to the posterior pharyngeal wall. Consequently, most cleft-palate patients possess, to a greater or lesser degree, a permanent rhinolalia aperta, or nasal voice, even after everything possible has been done both by operative and speech training measures.

2. The non-existence of the consonants *k* and *g*, due to lack of contact between the back of the tongue and the soft palate, a result of the postoperative inelasticity of the soft palate.

3. The non-existence or indefiniteness of the consonants *l*, *t* and *d*, on account of insufficient contact between the front of the tongue, the hard palate and the alveolar margin behind the front upper teeth; which is due to the fact that insufficient surface is presented to the tongue. Faulty dentition also aggravates this condition.

4. In harelip, difficulty in the production of the consonants *b*, *p*, *m* and *w*, because the tensed upper lip is not able to come in close contact with the lower lip.

The speech difficulties of these patients are nearly always increased by abnormal positions of the teeth and alveolar processes in relation to the body of the bones to which they are attached. These abnormalities must be taken care of before the best results in speech can be obtained.

Speaking of voice and speech results in these cases, I wish to call attention to a condition which these patients present —amusia. All cleft-palate patients suffer from amusia, a disturbance in the musical faculty. They all present one definite form of amusia, tone-deafness. Defects in the musical and speech faculties may coexist, or they may exist independently of each other. The independent occurrence of disturbances in the musical faculty points to the existence of a separate center presiding over the musical memory.

No progress can be made in the elimination of the nasality of cleft palate cases until the patient's musical sense has been developed to such a degree that he or she realizes the difference between nasal and non-nasal intonation. As soon as this takes place, the patient strives for purer intonation, and gradually the nasality diminishes, until to all intents and purposes it is completely lost. In other words, the patient has lost his tone-deafness and no longer suffers from amusia.

CONCLUSION

I have given only a brief survey of the subject, but I have tried to make clear the important relationship between the two specialties—dentistry and correction of defective speech.

Statistics show that more than 12,000,000 people, or 10 per cent of the population of the United States, have some sort of speech defect or voice abnormality. Of this number, 1,200,000, or about 1 per cent of our population, suffer from stuttering. But there are no statistics as to the percentage who suffer from mouth or jaw conditions in conjunction with their defect in speech. The reason for our lack of knowledge is the lack of cooperation between those who treat mouth conditions and those who treat speech conditions.

Every dentist or dental surgeon should have a general working knowledge of the physiology of speech and some of the speech anomalies due to mouth conditions. I hope that, in the future, the education of dental students will include a course in the physiology of speech and phonetics, and that there will be a better understanding between our two specialties. Through intelligent cooperation, we may reasonably look forward to the general betterment of mouth, voice and speech conditions. (Note: Several slides and charts were presented to illustrate the various oral anomalies discussed.)

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CARBON DIOXIDE, A RESPIRATORY STIMULANT IN NITROUS OXIDE-OXYGEN ANESTHESIA

By Lester A. Gerlach, D.D.S., Milwaukee, Wis.*

THERE has been much written on the relative merits of carbon dioxide as a respiratory stimulant when given in conjunction with nitrous oxide and oxygen for general anesthesia. Most of the gas machines today are equipped with rebreathing bags, their function being to collect the exhaled gases; namely, nitrous oxide, carbon dioxide and oxygen. The gases in the rebreathing bag are again mixed with the fresh gases from the tanks and inhaled through the nose-piece and mouth-piece. The purpose of the rebreathing bag is to save the unused portion of nitrous oxide exhaled for re-inhalation, and thus save gas. Another use for the rebreathing bag is to save the carbon dioxide which is exhaled, so that it also can be reinhaled and act as a respiratory stimulant. Some manufacturers of nitrous oxide-oxygen machines make a special carbon dioxide attachment, which is attached to the machine in such a manner that, at any time during the course of administration, the anesthetist can add a small amount of carbon dioxide to the mixture of nitrous oxide and oxygen.

In the practical administration of nitrous oxide-oxygen, one will note the even, regular respirations during the induction period, with the normal increase in number of respirations during the excitement stage, followed by even, regular respirations in deep anesthesia.

The rhythmic uniform breathing is characteristic of all normal administrations because the patient is instructed by the anesthetist to breathe easily and regularly when administration of the anesthetic is started. The even, regular respirations will continue for a short time during deep anesthesia, and quite suddenly the breathing will become irregular and somewhat spasmodic. This irregularity in respiration will disappear, only to reappear again in a very short time. These intermittent periods of irregularity in respiration will continue to appear throughout the anesthesia, and should the administration be of long duration, the periods of spasmodic breathing will occur oftener and be more pronounced. This description, of course, does not include cases of overdose. It only considers those cases in which the normal maintenance stage of anesthesia has been held, with no symptoms of overdose.

If, however, a small amount of carbon dioxide is added to the mixture of nitrous oxide and oxygen when the first irregularity in respiration occurs during deep anesthesia, the breathing will again become rhythmic, and there will be no recurrence of the spasmodic periods of breathing.

Why should this respiratory change take place and why does administration of carbon dioxide cause an instantaneous return to normal breathing? It is logically assumed that the carbon dioxide content of the blood has been diminished in some way and must be replenished in order to maintain normal respiration.

Carbon dioxide is acid in reaction and a certain amount of it is necessary to maintain the proper acid-alkali balance of the body. When the amount of

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carbon dioxide in the blood has been reduced, a definite alkalosis results, characterized by irregular spasmodic respirations.

What happens to the normal carbon dioxide ratio during nitrous oxide-oxygen anesthesia?

When the administration of the anesthetic is begun, the patient is given either 100 per cent nitrous oxide or 95 per cent nitrous oxide and 5 per cent oxygen. This means that the normal intake of oxygen has been greatly reduced, with nitrous oxide substituted. As inhalation proceeds, the mixture of nitrous oxide and oxygen is absorbed by the blood, until the saturation point is reached; at which time, there is deep anesthesia. With supersaturation, the stage of overdose is reached. That is why it is necessary to increase the amount of oxygen after the patient has been under the anesthetic for some time; *i. e.*, because the blood cells have become saturated with the anesthetic and it does not require so much nitrous oxide to maintain this saturation as it did to produce it originally.

During the normal course of respiration, the oxygen that is inhaled is absorbed by the blood and carried through the body. Carbon is a byproduct of metabolism in the body and its elimination is through the circulatory system. The oxygen, as it is carried through the body in the blood stream, unites with the carbon to form carbon dioxide, which, in turn, is eliminated by the lungs through exhalation.

During nitrous oxide-oxygen anesthesia, the amount of oxygen is so diminished that the normal formation of carbon dioxide does not take place. When the inhalation of the anesthetic is started, the amount of oxygen is such that carbon dioxide is being formed and eliminated. The reserve of oxygen in the blood is soon used up, and when the supply is diminished and very little is inhaled, the amount of carbon dioxide formed is greatly reduced. This diminution in the amount of carbon dioxide formed occurs gradually as the anesthetic is administered, and when the blood has become saturated with nitrous oxide, there is very little carbon dioxide formed. Consequently, the amount of carbon dioxide in the blood has been reduced to such an extent that the normal acid-alkali balance is disturbed. This would naturally produce the alkalosis and the irregularity in respiration.

When the amount of carbon dioxide formed is diminished, there is a lessening in the amount exhaled. When the gases are exhaled in the rebreathing bag, the amount of carbon dioxide is so small that when it is rebreathed, there is not enough to act as a respiratory stimulant. Therefore, it is necessary to add carbon dioxide to the gases administered in order to maintain normal rhythmic breathing. It is not necessary to administer carbon dioxide during the induction period, but when deep anesthesia is reached, the addition of a small amount of carbon dioxide will keep the respiration at the desired regularity.

CONCLUSION

1. Carbon dioxide is a respiratory stimulant in that it maintains the acid-alkali balance of the body.

2. The disturbance of normal metabolism during nitrous oxide-oxygen anesthesia is the logical explanation for the shortage of carbon dioxide. This lack of carbon dioxide is a natural occurrence when the amount of oxygen normally inspired is reduced.

3. The rebreathing bags will not contain enough carbon dioxide to act as a respiratory stimulant.

ETIOLOGIC FACTORS IN PERIODONTOCLASIA

By ARTHUR H. MERRITT, D.D.S., F.A.C.D., F.A.A.P., New York City

THERE is probably no phase of periodontia about which there is so much confusion as that dealing with the etiology of periodontoclasia. This is explained by (a) the number and complexities of the causative factors and (b) their interrelationship one with the other, (c) the fact that no two cases are ever exactly alike, (d) the possible combination of local and systemic causes and (e) the fact that there are still unknown factors to be taken into consideration. All this has a discouraging sound. It is little wonder that the rank and file of the profession are confused or that periodontoclasia is responsible for the loss of more teeth than any other dental disease, not because it is more common, but because it is less well understood.[1]

It may not, therefore, be inappropriate to reexamine the subject in the light of present knowledge with a view to a clearer understanding of what in reality may be regarded as the etiologic factors in periodontoclasia. When this is done, it will be found that our failure as a profession to control the diseases of the periodontium is not due to lack of knowledge, incomplete though that is, but to neglect in applying the knowledge already at hand, the application of which to the problems of daily practice would solve most of them.

One of the first requisites to a clearer understanding of our problem is the recognition of the fact that there are probably two types of periodontoclasia, one local and one systemic. Unfortunately, and as if to add to the confusion, the line is not clearly drawn between them. They are usually so interrelated that to make a differential diagnosis is always difficult and sometimes impossible. This is probably explained by the fact that, in both types, there are local and systemic factors in the etiology; that neither is wholly local or wholly systemic, and that whichever type develops will depend on which group of causative factors is dominant, the local or the systemic. And this in turn will be found to depend on several other factors which can be best understood by a study of the two types in their so-called pure form.

(Read before the Section on Periodontia at the Seventy-Ninth Annual Session of the American Dental Association, Atlantic City, N. J., July 13, 1937.)

THE SYSTEMIC TYPE OF PERIODONTOCLASIA

Gottlieb identifies a type of periodontoclasia characterized by a diffuse atrophy of the alveolar process which he believes to be primarily systemic in its origin. According to him, the cardinal symptoms are, in their order, loosening of the teeth, pocket formation and gingival inflammation. This, it may be said, is in exactly the reverse order of the so-called local type. The primary symptom in the systemic type is loosening or "wandering" of the teeth. One or more of the teeth may be involved in this migration. They may move in any direction except into their sockets. If they move into positions where they are subjected to excessive occlusal stress, they are said to be in "traumatic occlusion," though it cannot be too strongly emphasized that traumatic occlusion in these cases is not a primary etiologic factor, if indeed it is in any.

There are two theories, neither of which have been proved, to explain this type of periodontoclasia. The first is

Gottlieb's, which is that through some disturbance in cementoblastic activity, for reasons entirely unknown, no new cementum is laid down. On this point, he says: "The maintenance of a tooth in functional activity requires a continual deposition of new cementum on the surface of the root."[2] When this does not take place, the pericemental fibers lose their attachment to the cementum, thus withholding from the alveolar bone the stimulus of functional pull, causing it to atrophy; for it is Gottlieb's belief that it is only by the embedding of new fibers on the surface of the cementum that health in these tissues can be maintained.

The second theory is that of Broderick,[3] who believes that this type of periodontoclasia is brought about by some disturbance of the acid-base balance of the body as a result of which the tissue fluids are more alkaline than normal. It is his opinion that periodontoclasia and dental caries are antagonistic diseases and that this explains why it is that teeth relatively free from caries appear to be more susceptible to periodontal disturbance, a fact which every dentist has observed. It seems probable that, in most of these cases, the disturbance of the acid-base balance acts chiefly as a predisposing factor, which may be overcome largely by careful attention to local conditions. In other words, the local exciting factors in these, as in all cases, are the important factors. If these are controlled, one need not be greatly concerned about the systemic predisposition, though it must be admitted that such control is sometimes difficult. This imbalance in the acid-base equilibrium is believed to be brought about by such factors as heredity, errors in diet, activity and age.

In attempting to make a differential diagnosis in these cases, these are some of the things one should look for: relative youth in the patient, with teeth more or less free from caries, hygiene normal, teeth changing position, soft tissues firm and of good color, and if the condition is at all advanced, a fairly abundant discharge of pus. In other words, the condition is one that cannot be entirely explained by what is seen in the mouth.

LOCAL TYPE OF PERIODONTOCLASIA

The local type of periodontoclasia, which represents most of the cases seen in daily practice, presents quite a different picture. The average patient with this type is older. Susceptibility to caries, as shown by many fillings, is usually high, hygiene is none too good and the gums are apt to be soft and spongy. One does not need to look beyond the mouth to find the cause in this type of periodontoclasia. As a rule, there are several causes. First and foremost among these is oral sepsis, by which accumulation of tartar, bacteria and food débris is meant. Most important among local causes is the constant presence of bacteria, filling the gingival crevice and bringing about ulceration of its inner surface, resorption of the alveolar process, pocket formation and a chronic infection.

Since this type has its origin in the gingival tissues, there is no one thing more important in prevention and treatment than maintenance of these tissues in health. And of first importance in accomplishing this are cleanliness of the mouth and stimulation of the periodontal tissues. By these means, steps are taken to eliminate the two most important factors in periodontoclasia, bacteria and low tissue resistance.

A second etiologic factor is food impaction, which not only traumatizes the gingivae, but also encourages bacterial growth. Overhanging edges of fillings, partial prostheses, crowns, etc., all tend to injure the gingivae and increase their susceptibility to disease.

Excessive occlusal stress is another local causative factor. This may be brought about by malocclusion, interlocking of cusps, faulty operative procedures involving large inlays in supraclusion, crowns, bridges, partial plates, etc. Most

important of all perhaps is loss of the teeth. It is almost inevitable that when a number of teeth have been lost, those which remain will be subjected to undue stress and their supporting tissues broken down by the overload. In many instances, their replacement will only partially solve the problem; and since the prognosis in these cases depends in large measure on the establishment of functional occlusion, it will be evident that loss of the teeth is a serious matter in the prognosis and treatment of periodontoclasia. To sum up, then, it may be said that such conditions as disturbance of metabolism, oral sepsis, occlusal stress and food impaction are important factors in the etiology of periodontoclasia chiefly as they promote bacterial activity or lower tissue resistance. Or, to put in another way, if the bacterial flora of the mouth is kept within normal limits and tissue resistance is maintained at a high level through normal function or artificial stimulation, one need not be greatly concerned about the development of periodontoclasia, no matter what other etiologic factors may be present.

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RELATIONSHIP BETWEEN DENTAL AND CARDIOVASCULAR DISEASE

By James G. Carr, M.D., Chicago, Ill.

"Focal infection" is a term which belongs to modern medical phraseology, but the recognition of the connection between diseased teeth and disease of the body elsewhere has been alluded to occasionally for centuries. The first recognition of this relationship is attributed to Hippocrates, whom Kerr[1] mentions along with Rush, also quoting Dryden's complaint of rheumatism following the disease of his throat. Benjamin Rush also noted the connection between infections of the throat and rheumatism.

Thomson and Thomson[2] quote from Preston this story of ancient Assyria recorded by Professor Breasted:

Continually the King has been asking why Arad Nana, his physician, has not made clear his disease and has not cured him. Now Arad has sent a sealed letter which he hopes they will read before the King. He will make a prescription; let the ceremonies be carried out by a seer; let them bathe the King and straightway the fever will depart from the face of the King; let them apply to him oil two or three times; at once the strength of the King will revive; the King shall place on his neck the salve Arad will send him. On the appointed day let the King be anointed; Arad will speak the truth as the King demands; the pain in his head, his sides and his feet has come from his teeth and they must be extracted.

It is only within the present century that observations such as these have been followed by intensive scientific study and clinical application of the fundamentals of this mode of infection. To this type of infection, the term "focal infection" has

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been applied, and the theory has fairly dominated certain aspects of medical practice for more than two decades. That localized acute or subacute infections may be followed by general septicemia and pathologic change of various organs is undeniable. The opinion that persistent foci, latent or chronic, may be the source of infection elsewhere in the body is generally accepted. It has been accepted so widely and so dogmatically, in many instances, that the clinical applications of the theory need to be re-defined. For this statement, three reasons may be submitted: 1. Surgical treatment has been directed to the relief of symptoms without satisfactory demonstration of the connection between the disease, presumably secondary, and the focus of infection. 2. The various surgical procedures employed are not free from danger and the incidence of disastrous results therefrom is not inconsiderable. 3. The problem of multiple foci has often led to a veritable "surgical polypharmacy" without relief to the patient.

This is not the place for a discussion of the general application of the theory of focal infection: the tonsils, accessory nasal sinuses, the prostate, the gallbladder and other sites at which focal infection may develop are all regarded as possible sources of disease processes in other organs. Here, we are concerned with the dental aspects of the problem. This question may be put: To what extent do infections of the teeth and the roots thereof and peridental infections serve to produce and maintain acute and chronic heart disease?

This discussion is one of clinical experience and opinions based thereon. However precise the experimental basis may be, the clinical results are of the most importance and interest to the practitioner. A number of papers in the literature have been studied in order that the present status of the subject, as gathered from different opinions and experiences of various authors, may be shown.

In an address to dentists, Wyckoff[3] said:

A patient with chronic infection, such as that of a sinus, has impaired muscular efficiency generally; if he has any form of heart disease with diminished cardiac reserve, the chronic toxaemia throws an additional burden upon his heart. When chronic infections in these patients are cleared up, the patient bounces up like a rubber ball; his heart condition remains, but the added burden on it has been removed. Such relief occurs when chronically infected teeth from which toxins are steadily absorbed are removed. . . . Patients with degenerative heart disease are of interest for two reasons: they are very much better off if they do not have chronic mouth or other infections with their resulting intoxication; and they are likely to be a poor risk for dental operations.

Pardee, in his discussion of this paper, agreed that "dental infection may act to depress the function of an already damaged heart, without attacking the heart itself directly, but by depressing the body as a whole." He further stated that he had observed the development of subacute and acute heart infections following dental operations, particularly extractions of abscessed teeth.

Lintz[4] says:

There is a definite etiological relationship between infected teeth and cardiovascular disease; eradication of the focal dental infection and the proper immunization with autogenous vaccines leads to benefit in certain cardiovascular diseases. . . . In myocarditis and endocarditis focal dental infection plays a much larger rôle than heretofore supposed. Many of these patients can be restored to usefulness only by the eradication of infection combined with vaccine immunization.

He also discusses the relationship of various types of cardiovascular disease to the loss of teeth, stating that in 100 normal persons only 17.6 per cent of the teeth had been lost; whereas in seventy patients with cardiac disease, 42 per cent of the teeth had been lost. These figures are interpreted as indicating an excessive incidence of cardiac disease in persons with bad teeth. It would appear that the

reverse is equally likely to be true: the bad teeth may be the result of a poor diet and abnormal metabolic activity, the result of the cardiac disease.

Haden[5] has reported the results of intravenous injection into rabbits of bacteria from cases of chronic dental infection:

> The instances of lesions in animals after the intravenous injection of bacteria agree quite closely with the lesions due to chronic focal infection as observed in man." In another paper he remarks that "the tendency of bacteria recovered from active dental infection in patients suffering from heart disease of focal origin to cause valvular and myocardial lesions in the experimental animal is striking and adds evidence of a causal relation of the septic foci to the systemic lesion."

It is Faught's[6] opinion that "to fail to advise the removal of an infected area, when its removal does not jeopardize the life or future health of the individual, implies either negligence or lack of knowledge and experience." In his paper, he includes the case report of a man, who had previously had rheumatism, from whom nine teeth were removed at one sitting. This was followed within twenty-four hours by a chill and fever, and recovery only ensued after some months. He also cited cases to demonstrate (1) the importance of the complete removal of all foci of infection, and (2) the danger of multiple extraction of teeth.

Jones and Newsom,[7] in experimental work, produced dental abscesses in dogs:

> There were rather constant extra-cardiac structural changes. The hearts of inoculated dogs constantly showed very small vegetative or verrucous mitral and (or) aortic endocarditic lesions, patchy parenchymatous degeneration, nuclear changes, increase in the diameter of the muscle cell, and a slight round cell infiltration. . . . A positive relationship between experimentally produced focal dental infection and cardiac hypertrophy as measured by diameter of muscle cells was noted.

Lust and Spanier[8] report two cases of rheumatic fever in children at the ages of six and eight. The fever was of long duration. Prompt recovery occurred after removal of some bad teeth. One patient had a temperature of 104 degrees following the operation.

Antonius and Czepa[9] studied eighty-eight patients with various diseases to determine the significance of infectious processes in the production thereof. There were fifteen cases of chronic or subacute endocarditis in the group. In three of these, the teeth were removed. In one, there was no improvement. Thomson and Thomson[2] reiterate that all our observations clearly indicate the extremely important rôle played by the streptococci in periapical infections, and demonstrate, beyond doubt, the importance of dental sepsis as a source of systemic streptococcal infections as manifested in various forms of rheumatism, carditis, etc.

One author reports four cases of cardiac irregularity in which relief was obtained after the extraction of teeth. In the first case, in a man of forty, the heart was regular after three weeks and remained so for two years. In the second, in a man of sixty, while the removal of the teeth was followed by marked clinical improvement within six weeks, the irregularity showed no change for six months. It then steadily diminished in frequency and, two years later, the heart action became perfectly regular. In a third case, in a man of twenty-nine, the rhythm was bigeminal, the cardiac rate 80, the pulse 40. The patient complained of shortness of breath, early fatigue and precordial distress. Four teeth were removed and four months later the irregularity had diminished perhaps 30 per cent. A fifth tooth was then removed. "One year following the dental extractions, the heart action became perfectly normal."

In Case 4, a woman of thirty-five, with no other abnormality than a cardiac irregularity, had four teeth removed. Three months later, the rhythm was perfectly

regular. The first conclusion of the author is that "Heart irregularities will sometimes disappear when peridental abscesses are identified and surgically removed." Without disputing this statement, it is equally true that irregularities of this type are prone to come and go; such irregularities may persist for years without any detriment to the patient except his knowledge of them; we do not relate them regularly to pathologic hearts, and they are frequent in nervous persons, in those who use tobacco and coffee to excess and in persons past middle life. Though they may be associated with cardiac disease, the association is not constant. Furthermore, they are often missed by the physician in spite of the patient's insistence that they are present from time to time. Two of these four cases were stated to have returned to the regular rhythm one year or more after the removal of teeth; but even that is not proved unless the rate was repeatedly determined.

In the paper just referred to, there is cited a fifth case, in a man well along in years, with only occasional premature ventricular contractions, who declined to have his teeth removed:

> Four months later the premature systoles had disappeared, but in their place was substituted the absolutely complete irregularity of auricular fibrillation which, having once started in a person of his years, will likely become permanently established, despite any efforts at relief.

Such reports as these serve to make "confusion more confounded." We do not know that premature ventricular contractions are the result of myocardial disease due to disease of the teeth or myocardial irritability excited by toxins from infected teeth. Too many contributors to the literature have taken the same attitude toward this question, encouraging physicians to advise extraction on insufficient grounds, and the loss of teeth is not a negligible loss. In addition to the assumptions regarding the cause of disappearance of the premature contractions, it is further implied that a patient of rather uncertain years, probably an old man, suffered for his stubbornness by developing the absolutely irregular pulse, an auricular fibrillation. This type of irregularity is common in arteriosclerotic persons, in hyperthyroidism, in chronic mitral disease and in emphysema. It does occur occasionally in the course of infectious diseases, but is far from being common. It is most common in the later stages of chronic mitral disease. Arteriosclerosis is a degenerative disease, hyperthyroidism a metabolic disturbance, and the cardiac disease in emphysema may be due to mechanical conditions associated with prolonged right ventricular strain. What common factor precipitates auricular fibrillation we do not know; but, in the present state of our knowledge, we are not justified in attributing auricular fibrillation in the course of these diseases to infections of the myocardium.

Bridgeman[10] says that it is probably true that if one could examine all patients with premature systoles, it would be found that the majority of them would be relieved by giving up coffee and tobacco:

> Many times the patient's trouble is relieved by some simple hygienic measures such as the elimination of coffee and tobacco, by protection from mental strain or by the regulation of exercise, food, or habit of stool.

But he also insists that the presence of this irregularity places on the physician "the responsibility to prove, so far as it is possible, that there is no organic heart disease." He believes that such irregularities "are found equally frequently in normal and diseased hearts."

Price[11] says of this type of irregularity,

> They are to be found, for example, in persons with a history of rheumatic infection, the victims of tobacco poisoning, in digestive disturbances, super-normal blood pressure, neurasthenia, and as a result of the administration of certain drugs, such as digitalis.

According to Herman,[12] "In themselves

ectopies do not constitute evidence of heart disease and may be present in otherwise functionally normal hearts."

White[13] says, in his recently published book,

> There is no specific pathology of heart or nerves associated with premature beats. Organic heart disease may or may not be present. . . . Given a person with premature beats, the chances are more than ten to one that no significant structural abnormalities of the heart are discoverable. Premature beats are, however, relatively more numerous in the presence of heart disease than in its absence.

In relation to the association of cardiac disease with auricular fibrillation, White says,

> There is no pathology characteristic of auricular fibrillation. There may or may not be organic heart disease: usually there is. There may or may not be myocardial pathology other than hypertrophy: usually there is not.

He refers to the report of McEachern and Baker published in 1932, which stated that the chief etiologic relationship of various factors to auricular fibrillation in 575 consecutive cases was as follows: "Rheumatic heart disease, 34.4 per cent; coronary disease and old age, 31.1 per cent; hypertension, 16.9 per cent; thyrotoxicosis, 7.5 per cent; emphysema, 5 per cent; syphilis, 3 per cent, and miscellaneous, 2.1 per cent."

Reference may be made to another report which illustrates the proneness to connect cardiac disease with dental infection. The history begins with an operation on a suppurating left antrum, subsequently to which the teeth responsible for the antral disease were removed. Eight years later, a recurrence of the dental disease took place on the opposite jaw. Muscular rheumatism followed a year later and, during a later period of overwork, the patient developed a general exhaustion. At this time, the electrocardiogram revealed gross myocardial change, probably the result of coronary occlusion. Three years later, acute sternal pain occurred. Not long afterwards, the patient died of heart failure, eighteen years after the original mouth and antrum infection. The author obviously relates the coronary occlusion to the original dental and antral infections. The criticism may be offered that while the two conditions mentioned did coexist, this is no proof of any causal connection between them. Coronary occlusion has not been demonstrated to be of infectious origin; neither has it been demonstrated that surgical removal of dental foci of infection will prevent, delay or cure the arteriosclerotic process. To quote MacCallum[14]: "It appears that there is but little evidence in favor of the idea that infections, whether acute or chronic, play a great part in the pathogenesis of arteriosclerosis."

A different attitude is that of O'Rourke,[15] who cites McNevin and Vaughan:

> (1) Even if absolute proof of the existence of oral and other foci of infection cannot be submitted, it is nevertheless a reasonable assumption; (2) the strong circumstantial evidence advanced strengthens the assumption; and (3) the exact nature, however, of the effect which oral foci exert on the body in each case cannot always be determined. . . . If it be true that most diseases are due to a combination of forces, may it not be said that apparently harmless multiple foci constitute a force which though it may not be the most active, is nevertheless a needless burden to the individual.

MacCrae,[16] in his textbook, takes a conservative position. He writes thus of the treatment of focal infection:

> (1) Removal of the cause, the focus of infection. This demands proper diagnosis and should not be done until this is as definite as possible. A man with a mouth infection may have the real focus in his prostate. Caution should be exercised in the removal of teeth, especially in older persons. The mouth should be put in as clean a condition as possible and infected teeth removed at intervals. Wholesale removal of infected teeth may cause a severe reaction, or be followed by a general infection. Caution should

be exercised in the treatment of foci if the general symptoms are acute.

In Germany, divided opinions are found: Some agree essentially with the opinion of Pesch that "the removal of latent foci by dental procedures has had good results in the therapy of oral sepsis." H. Schlesinger,[17] on the other hand, regards "conservative measures as more correct than radical procedures."

To turn to a different aspect of the subject, attention may be directed to a report from Rushton[18] of two cases of subacute bacterial endocarditis with death following extraction of teeth. One patient was a boy of four, with chronic mitral disease. Rushton reviewed the cases of subacute bacterial endocarditis in Guy's Hospital during the years 1922-1927. There were forty such cases, four of which gave a history of extraction of septic teeth prior to the apparent onset of the disease.

Von Phul[19] notes the danger associated with the extraction of teeth, but believes that rigid surgical technic in such cases will generally prevent complications. He reports three cases of bacterial endocarditis following extraction of teeth in persons with chronic rheumatic disease and makes this further statement: "That such experiences are by no means uncommon is attested by Hartzell (personal communication), who has a list of ten deaths due to the extraction of badly diseased teeth or following very shortly thereafter." He also quotes Lucas (personal communication) that "such results do not occur provided the operator follows a definite complete chain of surgical asepsis in his cases."

Abrahamson,[20] after indicating his adherence to the current theory that infection must be eradicated, adds, "Yet the removal of septic teeth or tonsils in cases of chronic heart disease does not constitute quite so simple a problem as appears on the surface." He reports three cases of subacute endocarditis which supervened on removal of septic foci—twice in connection with teeth, once with the tonsils, "in circumstances which leave no doubt that the fatal issue was the result of removal of these foci." In one of these cases, a man of fifty-three with paroxysmal fibrillation failed to show any cause for the irregularity. Because he was not doing well, a cardiologist advised removal of two roots; the patient died within six weeks. In two of these cases, there were no symptoms or signs of endocarditis until the removal of the septic foci. The author thinks that "preliminary vaccination with an autogenous growth from the gums of teeth seems a rational procedure." Kapsinow[21] reported a case of acute pneumococcus endocarditis following extraction of teeth. The patient died about three weeks after the operation.

Blumer,[22] writing under the title "The Tragedies of Tooth Extraction," says: "It is not claimed that serious complications commonly follow the extraction of infected teeth." He reports four cases. Death occurred within twelve days to fourteen weeks after the operations. Only one of these cases was definitely cardiac in character. Apparently acute hepatic degeneration was the dominating disease in one instance, and nephritis in another. In one case characterized clinically by chills and high fever, necropsy was not obtained. Blumer discusses the different types of fatal lesion which may result from tooth infection:

1. Direct extension of the local inflammatory process with spreading inflammation, which may even invade the mediastinum, and death from toxemia.

2. Metastasis to neighboring tissues, such as occurs in cases of meningitis or thrombosis of the cerebral sinuses following tooth extraction.

3. General septicemia or septicopyemia, which may terminate in:

(a) Acute septicemia with or without endocarditis.

(b) Chronic septicemia with degenerative processes in the internal organs.

(c) Prolonged septicopyemia with the formation of abscesses.

Gerstley[23] reports a case of death from bacterial endocarditis in a boy of thirteen with rheumatic fever and mitral disease. One tooth was removed at the first operation; a second, later. The author says, "The clinical observations on this boy again confirm the clinical experience of many men, that minor operative procedures, particularly when foci or infection are supposedly present, may be dangerous."

Mitchell Bernstein[24] reports the death of a man of twenty-five from subacute bacterial endocarditis following the extraction of two abscessed teeth. This patient had an old mitral lesion, not active at the time of the extraction.

Vanderhoof and Davis[25] report two cases of subacute bacterial endocarditis with death following the extraction of teeth, one in a man of fifty-six with an old rheumatic lesion and one in a woman of forty-eight with no prior cardiac disease. They introduce their paper thus:

> During the past few years we have been impressed by the number of serious complications which have followed relatively simple operative procedures in the oral cavity, the throat and the upper respiratory tract.

Herbert H. Brown[26] reports one case "in which chronic infective endocarditis followed immediately upon extraction of a tooth." He suggests the use of vaccines before removal. This suggestion has been made by others, as has been noted. In view of the uncertainty and unreliability of streptococcus vaccines in general, for prophylactic purposes, reliance on such vaccines to protect the patient who is to have a tooth extracted is not warranted. Brown quotes Sir William Wilcox thus: "I have, on several occasions, seen fatal septicemia and malignant endocarditis follow extraction of infected teeth in a patient in poor health with a low immunity."

Musser[27] reports the case of an old gentleman who died from an acute endocarditis the symptoms of which came on about one week after the extraction of half a dozen infected teeth. He goes on to say: "Under any circumstances the many reported occurrences of the disease after measures to remove foci of infection indicate that extreme precautions should be taken when such foci are eradicated by surgical procedures in patients with pre-existing valvular disease."

Dexter[28] reported twelve cases of sepsis after extraction. In only one did subacute bacterial endocarditis develop. After stating that "there is scarcely a physician here present who has not seen one or more instances of almost miraculous amelioration of the symptoms of some inflammatory process following the removal of infected teeth," he adds, "That extraction of teeth may lead to extremely grave or even fatal local or general infections is a fact that I think has been lost sight of in the general wave of enthusiasm for their removal."

If we were sure that removal of infected teeth would eliminate the danger of primary endocarditis or prevent the development of myocardial fibrosis; if such a procedure would protect the patient with chronic valvular disease from further attacks or promote the recovery of acute inflammatory disease, occasional accidents might be discounted as inevitable accompaniments of a procedure which, in general, is of great value. But such a theory cannot be supported. Rarely are we able to connect the onset of acute rheumatic fever or a recrudescence of old mitral disease with an infected tooth, if the tooth is left alone.

TONSILLECTOMY

Since tonsillectomy to prevent cardiac infection is based upon the theory of focal infection, even as dental extractions are, it is permissible to refer to this procedure. Warburg,[29] in an extensive article, states:

> It is hardly to be questioned that tonsillectomy does not give noticeable protection

against recurrences and myocarditis; it is also questionable whether tonsillitis provides significantly against first infections. (It has been shown that a very large number of first attacks in those whose tonsils had been removed have been encountered in New York and London.)

He quotes Kaiser, who found as the result of the operation "only a slight protection against rheumatism, doubtful protection against myocarditis, and cases of chorea more frequently in the operated than in the non-operated." Warburg adds: "We cannot place great hopes upon the war against the tonsils."

Ingermann and Wilson (cited by Kaiser) found among eighty-eight children between one and eleven years of age, exactly as many rheumatic conditions as before the tonsillectomy. T. Ducket Jones believes that the presumed efficacy of tonsillectomy is more likely to be shown in young children. As the children reach the middle of the second decade of life, tonsillectomy is apparently more efficacious because the patients have reached an age of relative immunity to the rheumatic virus.

Recently, S. J. Lang made a statement in the course of a demonstration at the Evanston Hospital Clinical Conference which is very significant. He presented roentgenographic films of various local disturbances of the back and the sacroiliac regions. These were degenerative, not infectious processes. In the course of his talk, he said that he had been led to these studies because of repeated failure to relieve these patients of pain by the removal of foci of infection. He turned his studies in a new direction; found degenerative processes with disability and pain-producing qualities, and by mechanical measures gave the patients relief. He added that sacro-iliac arthritis has not been demonstrated in any one of several score of cases.

Arnett and Ennis[30] presented data almost exclusively from young people:

> While these data offer no support for the view that dental infection causes rheumatism, chorea, valvular heart disease, or electrocardiographic abnormalities in these vigorous young college students, yet, if the body resistance should be lowered by exposure, hardship or advancing years, it is possible that such might be the case.

They believe that granulomata and other dental infections may cause trouble in later years. "It would seem the part of wisdom, therefore, to treat these dental infections as potentially dangerous. This can often be done without the extraction of the affected teeth."

The wholesale removal of devitalized teeth and teeth with granulomata is certainly without justification in healthy young persons such as comprise the present study material.

Some mention should be made of a type of disease which has been responsible for much fruitless surgical procedure of major character. There are certain persons of a particular body type, with a long narrow body and the so-called "hanging heart," an unstable pulse and often a low blood pressure. Vasomotor disturbances are frequent. These patients are easily fatigued, not capable of hard work and often inclined to faint. Exercise notably increases the cardiac rate. Often, the symptoms are interpreted as cardiac. The easy fatigability, the tendency to faintness, the unstable pulse, especially rapid after exercise, the subjective symptoms of cardiac pain and the tumultuous action of the heart may lead to a diagnosis of cardiac disease. These symptoms have their basis in the fundamental physical and nervous structure of the patient. If the teeth require surgical measures, they should be taken; but attention to the teeth will not alter the patient's constitution nor relieve him of the cardiac symptoms, which are often troublesome.

It appears that the time is ripe to reappraise our clinical experiences with the application of the theory of focal infection to practice, and to revise in the direction of greater conservatism our clinical procedures. There has been a disposition

to accept the final validity of the focal infection theory, and to apply it more widely than is justifiable. Moreover, the unfavorable or harmful results following the surgical treatment of such foci, especially in connection with cardiac disease, are events of sufficient importance to impose on us the duty of caution. The theory has tended to become a dogma to both the profession and the laity, a "mass psychosis" as one German author has put it. It is obvious that radical thoroughgoing application of the theory to practice is fraught with danger. Our practice should be modified in the direction of restraint, the establishment of more accurate indications for surgical measures, due recognition of the hazards and, perhaps, better surgical technic. We must recognize that the strict application of the theory of focal infection to practice has its dangerous aspects.

Demonstration of a relationship between foci of infection and arteriosclerosis has not been forthcoming. Hypertension is not an infective process. Various infections, such as typhoid and influenza, pneumonia and general septicemia, are accompanied by acute myocarditis which may develop into the chronic form, though this sequence is not accepted as a common event. Diphtheria produces an acute myocardial degeneration, and other infections may cause myocarditis. The relationship of these conditions to chronic myocarditis is not well explained. In general, chronic myocarditis, as we see it clinically, apart from the rheumatic group, is degenerative in origin, the result or the accompaniment of arteriosclerosis, with or without hypertension. Coronary disease is a part of the arteriosclerotic process. Cardiac irregularities may occur in the course of any one of these groups, though the syphilitic type is rather conspicuously free from them. Treatment is symptomatic. Often, certain types of irregularity occur in the absence of demonstrable disease. The treatment of these does not include dental surgery. Auricular fibrillation, paroxysmal tachycardia and heart block are types of abnormal rhythm which call for careful appraisal when dental surgical operations are necessary.

Necessary dental work for local conditions may be employed without fear in the presence of compensated cardiac disease. It is wise to confine the dental procedures to necessary procedures and to refuse to undertake much work at one time. If congestive failure is present, only that work should be done which is deemed imperative; in the presence of angina or known coronary disease or aneurism, the combination of vaso-constrictor drugs, such as adrenalin, with the local anesthetic should be discountenanced or employed with care. Abrupt rise of pressure due to adrenalin may precipitate an anginal attack. Dental work may be undertaken to improve the nutritional state with more circumspection; dental surgery directed to improvement of the cardiac state by the removal of infected teeth, to correct cardiac irregularities or to lessen the tendency to recurrent infections, is not indicated. Dental surgery to cure or relieve cardiac infection is contraindicated.

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DIAGNOSIS AND TREATMENT OF NEUTROCLUSION CASES

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THERE are varied and diverse views concerning the classification of malocclusion, and my first purpose will be to consider the intrinsic factors which must be a guide in arriving at a decision as to the grouping of the various cases.

Many efforts have been made to formulate a classification which would obtain universal acceptance, but that ideal classification has not yet been developed. This is due chiefly to the varied opinions concerning the etiology, of which many earnest workers say practically nothing is known. Nevertheless, each of us, in his effort to make a diagnosis, must classify his cases upon some theory.

About the end of the last century, Dr. Angle brought considerable order out of chaos when he presented what appeared at the time to be an ideal classification. His terminology was so distinct that, on the basis of his assumption of the constancy of the maxillary first molars in taking their normal mesiodistal positions in the dental arch, the various deviations that presented themselves automatically classified each case in the proper group. However, the weak points in this classification gradually became evident, athough a few of the more ardent adherents still seem to believe that it is sound. Dr. Angle was, undoubtedly, one of the foremost men of his time in the teaching and practice of orthodontics, but we must realize that no person is beyond the possibility of error. It was my good fortune, twenty-nine years ago, to receive training in Dr. Angle's school, and I believe that the excellence of his clinical teaching has never been surpassed. He also developed numerous orthodontic mechanisms the clinical manipulation of which raised very markedly the standard of orthodontic treatment.

The chief weakness in the Angle classification is its failure to relate the denture to the cranium definitely, but on this point Angle was not alone. Simon, with his "law of the canines," sought to correct this, but, owing to anatomic variations, his "orbital plane" is not sufficiently reliable to be dependable; and so his scheme does not satisfactorily overcome the difficulty. Broadbent is working out a plan which promises well, but is yet to be proved, and requires complicated and expensive apparatus. I believe that this problem is not so difficult as we have thought it to be, and that a very simple procedure will aid us in determining this relationship if the operator has sufficient knowledge of facial harmony. The plan which I have followed for some years, and have outlined in previous contributions, has been criticized as being dependent on individual judgment and lacking in scientific background. However, until there is established more dependable anatomic landmarks from which to obtain measurements, we must rely on our judgment born of experience.

In order to diagnose a case of malocclusion, we must formulate some theory to account for the anomaly which we seek to correct. If our analysis is based upon

(Read before the Section on Orthodontia at the Seventy-Third Annual Midwinter Clinic of the Chicago Dental Society, February 16, 1937.)

a theory which is not sound, we have erred in our very first step, and this error leads on to others, finally resulting more or less in failure. The crux of this problem is the uncertainty of the etiology.

The methods in practice of Edward H. Angle probably gained wider acceptance in America, if not in the world, than did those of any other author or teacher. Any one familiar with his latest contributions, however, could not fail to recognize that he changed his views quite definitely on at least one important theory as compared with those which he held when the last edition of his book was published, in 1907. On this point, particularly, I shall ask your consideration. At that time, Dr. Angle taught that molars and premolars were rarely ever forward of their normal relation to the cranium, except when individual teeth had tipped forward as a result of the loss of teeth anterior to them. To my mind, this was Dr. Angle's greatest error. He later corrected it, but not until many of his followers had become so inured to the old idea that they either failed to recognize his altered opinion or did not endeavor to develop the requisite technical skill to manipulate the more intricate appliances necessary to carry out the distal movement of teeth in both dental arches.

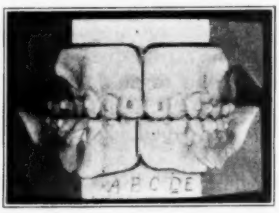

Fig. 1.—Lateral displacement of mandible to the right side.

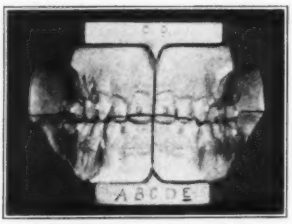

Fig. 2.—Casts shown in Fig. 1, with mandible biting true on center.

In a paper[1] introducing the edgewise arch mechanism, Dr. Angle speaks of "the law which must be observed in detail if treatment of an unbalanced denture—malocclusion—is to be successful." In this presentation, he stipulated three details, one of which reads as follows:

Third, and quite as important as the other two, though seeming as yet to be little appreciated, there must be established fully normal upright axial positions and relations of the teeth, if the teeth are to balance with the muscles and sustain and normally maintain the great weight of occlusion.

To make this last point clear: You all know that in addition to the usual irregular cusp relationships, there is found in a very large percentage of cases of malocclusion a more or less abnormal tipping or leaning forward of many or all of the teeth on either or both sides of one or both dental arches, and often, also a slightly bodily drifting forward of their normal positions. These conditions may, of course, be found in cases of all classes, divisions and subdivisions of malocclusion of the teeth.

The edgewise arch mechanism has great possibilities in the hands of those skilled in its manipulation, particularly in correcting the abnormal angle of inclination of the roots of the teeth, but there are many good technicians who doubt the possibility of carrying teeth back bodily with this appliance without the use of extra-oral anchorage, which, however, Dr. Angle advised the less experienced operators to employ.

Fifteen or more years ago, I began an intensive search for the cause of our numerous failures, and any who are sufficiently interested may refer to some of my contributions,[2] and follow the trend of this investigation up to and beyond the point of my acceptance and application

in practice of the "forward translation theory" of J. Sim Wallace. The basis of this theory originated 165 years ago with John Hunter's description of the growth of the jaws, quoted in J. Sim Wallace's work,[3] the essence of which is that growth takes place occipitally, and that the permanent teeth do not normally move forward into positions in the dental arch originally occupied by the deciduous denture.

It is not necessary to cover again the details of this theory, with which many are probably familiar or which may be found outlined in Sim Wallace's book, as well as in some of my papers referred to above. However, there is an important detailed technic leading up to the analysis and classification of a case of malocclusion which must be covered. As this technic was discussed fully in a paper[4] read before the Great Lakes Association of Orthodontists, I shall repeat here only the portion of that paper covering these points.

Our first step, when undertaking the correction of a case of malocclusion, is to obtain accurate impressions of the dental arches. Before inserting the plaster for the maxillary impression, a line about one-eighth inch long should be drawn with an indelible pencil over the median raphe just about opposite the first permanent molars. This mark will appear on the impression, at which point a slight groove should be made. This will stand out as a ridge upon the cast. We thus have a record of the median line of the palate, which may later be connected with the median line of the face marked on the labial surface of the cast. Other essentials are full-mouth roentgenograms and full-face and profile photographs (or roentgenograms of the head) of the patient. There are other points which must be observed and marked, in order that a careful analysis of the case may be made and a definite plan of procedure formulated. At this stage, accurate technical detail is most important, and if it is neglected, an endless series of difficulties may result.

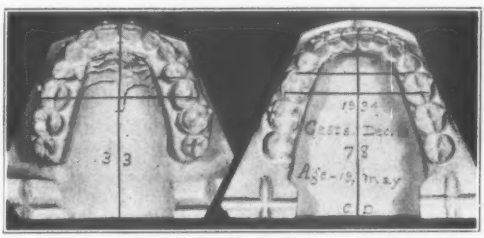

Fig. 3.—Same casts as in Fig. 2, showing median line on each, also locking grooves on posterior portion of each cast.

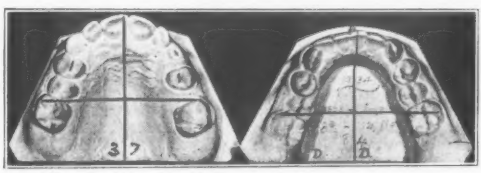

Fig. 4.—The transverse line registers the axial relations, and here shows greater forward displacement of both maxillary and mandibular teeth on the right side than on the left.

The vertical plane of the head must be established, and many devices have been suggested to obtain this, one of the most scientific and reliable of which is prob-

ably that being worked out by Broadbent. So far, my own has been a very simple one, and it has been criticized as being unscientific. However, as this feature, as well as the etiology, has not yet been settled, individual judgment must still come into play. The profile picture is the more important one, and a careful study of the dental arches and face of the patient will enable one with experience to mark upon the photograph this plane, which Angle at one time called the "line of harmony." We now decide the type of face and also determine whether the mandible is normal in its horizontal growth.

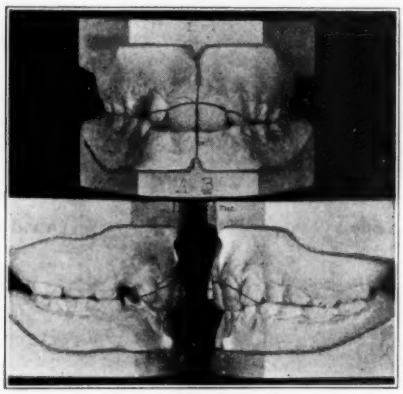

Fig. 5.—Shows resting line of upper lip marked on maxillary cast, also excessive downward growth of premaxillae. Patient eleven and one-half years of age.

In a face of the straight type, with a normal mandible, the line of harmony should touch the glabella and the anterior part of the chin. In one of the mental eminence type, with mandible normal, the mental eminence would extend slightly forward of the line of harmony, the amount being governed by the prominence of the eminence. A profile photograph without some record of the perpendicular line of the head is practically worthless. The median vertical line of the face is also determined at this time, and this should be marked accurately on the labial surface of the maxillary cast.

It is now that we decide whether there is any lateral shift of the mandible, and the median line of the latter is marked on the mandibular cast. This is all done before the casts are trimmed, and to establish a guide for the trimming. The resting lip lines are traced approximately on the casts at this time, and the latter are now ready for the laboratory technic. The bottom of the base and top of the capitol are trimmed first, and the median line, traced on the labial surface of the maxillary cast, is now extended over the palatal surface of this cast and connected with the point marked over the median raphe opposite the molars. The posterior surface of this cast is trimmed at a right angle to this line, and the line extended on this surface.

The posterior surface of the mandibular cast is now trimmed parallel to that of the maxillary cast. If the mandible is true to center, the median line marked on the labial surface of both casts will form a continuous line. The median line of the posterior surface of the mandibular cast may now be obtained by extending on this cast the line previously inscribed on the posterior surface of the maxillary cast. This may then be connected on the floor of the mandibular cast with that on its anterior surface. We thus have the median line on the occlusal surface of both casts, a detail of extreme importance. If there is a lateral shift of the mandible, as shown by the vertical lines on the labial surface of the casts in Figure 1, the median line at the posterior portion of the mandibular cast cannot be obtained so simply. To overcome this difficulty, the patient, looking in a mirror, practices biting with the mandible true to center, and then, with compound, a bite is obtained in this position with the material so placed that it will not hide the anterior teeth.

With this condition present, it is best

to have an extra set of casts, and, on the back of one maxillary cast, to build an extension and cut grooves on the occlusal surface to form a locking surface. Now this and the mandibular cast are placed in the bite and the mandibular cast is extended to harmonize with the extended portion of the maxillary cast. We now have an extra set of casts, as if on an articulator, with the mandible biting true on center, as shown in Figure 2. The median line of the posterior portion of this mandibular cast may be gotten in the regular way. Figure 3 shows the occlusal view of such a set of casts.

Another important feature in the marking of casts is the transverse line, which is drawn at a right angle to the median line, and placed at the contact point between the second bicuspid and first molar on the more distal side, if there is any difference in the forward displacement on either side. This registers a permanent record of the relative axial relations on the two sides, as shown in Figure 4, where there is a greater forward displacement of the teeth on the right side than on the left in both arches. These transverse lines correspond with those which I have on my Hawley charts. It is needless to discuss further the value of these landmarks. The rest of the detail of trimming the casts is familiar to all.

With a case carefully mapped out, as outlined above, we are now able to make a correct analysis very quickly. But let me here stress the point that every step up to this stage must be taken with the greatest care, since a mistake, or neglect in any detail, in this preliminary work will engender difficulties which may not become apparent until further errors have inevitably crept in, correction of which may be very difficult.

Now, let us consider classification in detail, and see wherein some of the systems seem to have been in error. Angle divided all cases into three distinct groups, Classes I, II and III, with divisions and subdivisions. He said that those in the second class usually showed "underdevelopment" of the mandible, but he did not suggest any differentiation in treatment of those cases with a normal mandible and those in which the mandible was "underdeveloped." Lischer's classification[5] was somewhat similar to that of Angle, but with a different, though clearly descriptive, terminology. He, however, made a distinction between those cases with a normal, or nearly normal mandible and those presenting "extreme malformation of the jaws," some of which he said, would, in time, undoubtedly be recognized as "micro-mandibular or mandibular retroversion deformities." Lischer also stated that the distoclusion in these extreme cases is "merely a symptom," and spoke of the supposed distal position of the teeth in the mandible as constituting a "true distoclusion."

Fig. 6.—Same as Fig. 5, showing loss of both right deciduous canines, and shift of mandibular incisors to the right side.

In my opinion, very rarely is there a distal position of the teeth in relation to the bone in which they are placed. For years, I have been endeavoring to show that misconception on this point has been

responsible for many failures in treatment.

Classification should be based upon the relation of the jaws to the cranium, not upon the relation of the teeth in the mandible to those in the maxillae. In an effort to clarify this point, let us examine a few cases, in order to ascertain what constitutes a case in neutroclusion.

Figure 5 shows the casts of a boy 11½ years of age. There is extreme excessive vertical growth of the premaxillae, as well as some increase in the vertical height of the mandibular incisor region, with an overbite of 8 mm. The buccal teeth are

Fig. 7.—Profile of patient whose casts appear in Figs. 5 and 6, showing the "line of harmony" and symmetry in the vertical height of the different segments of the face.

very nearly in normal mesiodistal relation; but until we see the boy's face, we do not yet know whether this is a neutroclusion case or not. The mandible is true to center.

Figure 6 shows the occlusal view. The maxillary and mandibular right deciduous cuspids have been lost and practically all the space for the mandibular cuspid has closed and the incisors have tipped to the right. A little of the space for the maxillary cuspid has also closed and, as a result, there is slightly more forward translation of the maxillary molars on the right side than on the left. The axial relations of the mandibular molars are almost symmetrical, notwithstanding the fact that the deciduous cuspid on the right has been lost.

Figure 7 shows a profile photograph of the boy. His is the mental eminence type with mandible practically normal. This is a neutroclusion case, presenting complications in treatment which we shall not attempt to discuss in this presentation.

Figure 8 shows the casts of another case, in a girl 12½ years of age. As the

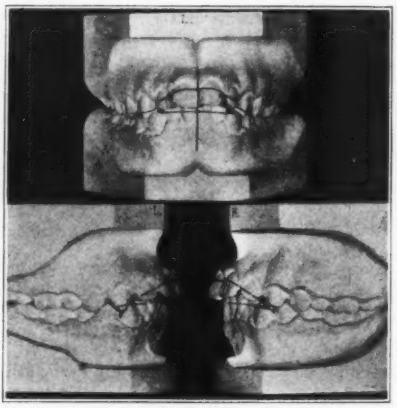

Fig. 8.—Casts of a girl twelve and one-half years of age. Classification from cusp relationship alone would place this case in the neutroclusion group.

mesiodistal relations of the teeth are normal, this, according to the general conception, would also be classed as a neutroclusion case. Figure 9 presents the occlusal view of these casts, and shows, according to our landmarks, that the axial relations all around are symmetrical. Figure 10 shows protographs of the patient in which there is definite evidence of a maxillary protrusion and shortening of the mandible. A forward translation of the maxillary teeth, with normal interdigitation of their cusps when in occlu-

sion, definitely indicates also forward displacement of the mandibular teeth, unless there was abnormal horizontal growth of the mandible. In this case, the crowns of the maxillary incisors are tipped forward and these teeth are spaced. If there were a normal mandible, this would be a neutroclusion case; but as the mandible is short, the forward drift of the mandibular teeth is greater than that of the maxillary teeth. Now, if we picture the mandibular teeth as back in normal relation to the mandible or to the apical base, we have a distoclusion case, the proper correction of which would be brought about by increased horizontal growth of the mandible, the procedure to obtain which I have outlined in former papers.

On page 129 in Simon's book,[6] are casts of a somewhat similar case, shown here in Figure 11, except that the crowns of the maxillary incisors in this case are retruded, as compared with the root ends. Both pictures here are of the same casts, but on the one on the right I have traced, as nearly as possible, the axial lines of the roots of the bicuspids and cuspid, the line over the cuspid being extended to the top of the cast in order to compare the long axis of this tooth with the orbital plane marked on the cast. There appears to be a forward translation of the roots of the maxillary teeth, the crowns being held back by the locking of their cusps with those of the mandibular teeth. Figure 12 shows the profile of this patient. To explain the incongruity of normal mesiodistal locking of the cusps in this patient with a short mandible, Simon offers the suggestion of "an individual family type." To my mind, this is also a distoclusion case.

Thus, these cases belong to the same class, differing only in some details. In my case, the crowns of the maxillary incisors are tipped forward definitely and those of the buccal teeth somewhat, a Class II, first division (Angle), the patient probably a mouth-breather. In Simon's case, the roots of all the maxillary teeth apparently are translated forward, with retruding incisors, in his terminology a "maxillary alveolar protraction plus anterior dental retraction," and a Class II, second division (Angle), in a normal breather. In my opinion, the cases are equally amenable to treatment.

Figure 13 shows the casts of a girl seventeen years of age, with a normal mesiodistal relation of the teeth. The median line of the face, marked on the casts, shows a shift of the teeth to the right, in the mandible very definitely so. Figure 14 shows the occlusal view. The mandibular first bicuspid and cuspid are much farther forward on the left side than on the right, owing to the late eruption of the right second bicuspid. In this case, there is a double protrusion associated with slight shortening of the mandible, as shown in Figure 15. With so much forward translation of the teeth in both arches, this case could not be successfully corrected without the removal of four teeth; nor could facial harmony be established without this procedure as well as some increased horizontal growth

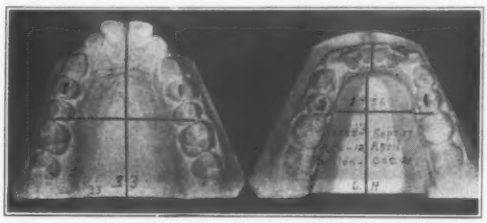

Fig. 9.—Same as Fig. 8. Axial relations symmetrical.

of the mandible. Probably this case also belongs in the distoclusion group.

In an effort to overcome the difficulty in arriving at an analysis of a case, I would suggest a tentative outline of still another classification of dental anomalies, composed of four distinct groups, based upon the relation of the jaws to the face and cranium, as follows:

1. All those cases in which there is normal horizontal growth of both maxillae and mandible.

2. Cases with normal horizontal growth of the mandible and lack of such growth in the maxillae. (Very few cases would fall in this group.)

3. Those cases in which there is lack of normal horizontal growth of the mandible.

4. Those cases with excessive horizontal growth of the mandible, included in which would be certain cases showing a lack of normal horizontal growth of the maxillae.

These groups should be further subdivided, in order to fully cover various malrelations of individual teeth, or groups of teeth, to the normal plane of occlusion.

Some may assert that, in this suggested grouping, there is no material change; but there is. Two cases of my own just shown (Figures 8, 9, 10 and 13, 14 and 15), with normal mesiodistal locking of the cusps, would be classified by most orthodontists as belonging to Angle's Class I, or neutroclusion, notwithstanding the fact that in both the mandible is short. Again, there are cases in which there is a definite forward displacement of the maxillary teeth, as evidenced by the facial lines, as well as by the casts, with a so-called distoclusion of the mandibular teeth, but with a normal mandible. The latter cases are habitually placed in Class II, or distoclusion, when in reality they belong in Class I, or neutroclusion.

We also have cases, with a normal mandible, presenting a forward translation of the maxillary teeth on both sides and of the mandibular teeth on one side, to the extent that there is a distal occlusion of the mandibular teeth on the opposite side. A case presenting these deviations would ordinarily be classified as a so-called unilateral distoclusion, or the subdivision of Class II, Angle. It is nothing of the kind, being a Class I, or neutroclusion, case. The same unilateral condition may be present in a case with a short mandible, in which instance we would have a distoclusion case due to lack of growth of the mandible. With a classification based upon the forward translation theory, and the relation of the jaws to the cranium, there is none of this complexity.

In the classifications which I have criticized, this error of the so-called "unilat-

Fig. 10.—Photographs of patient whose casts appear in Figs 8 and 9, showing a short mandible, placing this case in the distoclusion group.

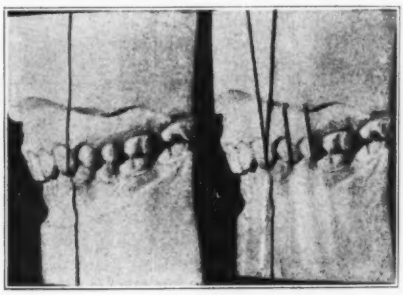

Fig. 11.—Casts from Simon's book of a case with similar cusp relationship as that in Fig. 8.

eral distoclusion" is common to all. Practically all of us now agree that teeth drift forward of their normal relation to the apical base, in some instances the forward displacement being greater on one side than on the other. Then why say that the teeth on the more distal side are in "distal occlusion"? In the correction of these cases, the procedure of most operators has been to carry the teeth on the more distal side forward. For years, I have been trying to stress the magnitude of this error.

Let us look again at Figure 14—the occlusal view of the casts of the case in which there is a double protrusion. The transverse lines show that there is no more forward displacement of the molar teeth on one side than on the other in both arches; but, on the right side of the mandibular arch, the second bicuspid is crowded lingually and more than half of its space is closed. As indicated by the median line on this cast, the incisors have been forced to the right to the extent of more than half the width of the central incisor.

In Figure 13, the buccal view of these casts, there is, on the left side, normal mesiodistal locking of the cusps of all the buccal teeth; whereas, on the right side, the mandibular first bicuspid is locked in a distoclusal relation with the first bicuspid in the maxillary arch. Would it be sane to carry the mandibular right first bicuspid, cuspid and incisors on this side forward, and thus cause greater protrusion of the anterior teeth? Measurements show that the dental arches in this case are only very slightly under normal width. Many operators, in their efforts to get all the teeth into harmonious arch form, where there is crowding, expand the dental arches too much, and carry the incisors too far forward. Later, they wonder why their cases do not hold or why recession takes place. This last point was covered fully in a former paper.[7]

Fig. 12.—Photograph of patient whose casts appear in Fig. 11. Not a neutroclusion, but a distoclusion case.

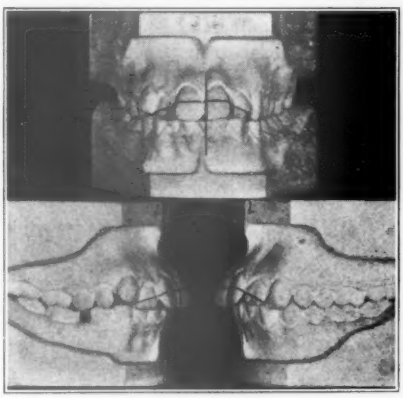

Fig. 13.—Casts of a girl seventeen years of age. Normal mesiodistal locking of molars.

I must again declare my belief that malocclusion is the result of a forward translation of the teeth, due to lack of normal backward growth of the jaws. This theory of Sim Wallace is constantly gaining new adherents, and will some day be accepted as an established scientific fact. With this theory as the basis for diagnosis, the complexity of the majority of our cases becomes very much lessened and their analysis simplified. Treatment, also, where skilfully applied, obviates the use of retaining appliances in most in-

stances. Why the whole orthodontic profession does not realize this fact is beyond my comprehension. The diagnosis and treatment of my own cases have been based upon this theory for the past ten years, and the results have been a revelation.

I presume that many of you are engaged in general practice, and I have failed to suggest any procedures which might be expected to bring successful results easily. If I have succeeded in guiding the less experienced away from disastrous mistakes, I will have done all a greater service. Too much has been claimed for the efficiency of simple appliances, such as the lingual wire and biteplates, and much grief has resulted, not only to general practitioners who endeavor to practice some orthodontia, but also to many specialists. The lingual wire has its place, but it is not an efficient mechanism for the correction of complicated cases of malocclusion where mesiodistal and vertical movements of the teeth are required.

General practitioners who send to dental (or supposedly orthodontic) laboratories casts upon which to have appliances constructed are in the position of purchasers of "wild-cat" mining and oil stocks: they very often are left "holding the bag." If a practitioner cannot construct the appliance necessary for the correction of a case of malocclusion, what chance does he have to manipulate such apparatus successfully? The construction of an orthodontic mechanism is much more simple than its manipulation in practice. For those with limited experience, attempts to correct, with commercial laboratory mechanisms, any other than the most simple cases are very apt to result in failure.

In considering the treatment of neutroclusion, all cases exhibiting macrognathia or micrognathia must be eliminated, as the latter present conditions apart from the mere mesiodistal relations of the teeth in the dental arches, and so must be placed in another class. In the correction of simple neutroclusion cases, not complicated with excessive overbite, there would be little to do except to bring all the teeth into the "line of occlusion," and carry distally any molars which had drifted forward slightly as a result of premature loss of deciduous teeth. In some instances, this might be accomplished with simple appliances. Unfortunately, in many cases, there is more or less forward translation of some or all of the teeth in one or both jaws, requiring an efficient mechanism and exacting technic if we are to assure a highly successful result, as greater anchorage becomes necessary.

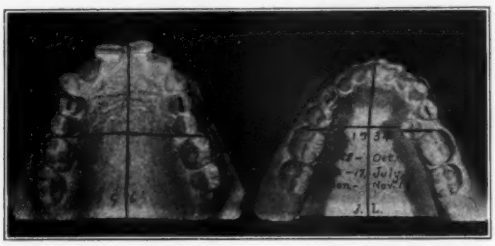

Fig. 14.—Occlusal view of casts in Fig. 13. Note partial closure of space for mandibular right second bicuspid, and consequent lateral shift of incisors.

The most difficult problem in the preparation of this paper was how to present the subject of treatment. There is great variation in conditions in cases in this class, and it is possible to consider these in only a general way. After careful consideration, I decided to present

an individual case, illustrating as clearly as possible the important factors which must be taken into consideration, so that a true picture may be had of what has taken place in the denture, and thus a definite procedure be formulated to correct the anomaly.

Figure 16 shows the casts of a mixed denture, in a boy nine years of age. All permanent incisors have erupted except the maxillary lateral incisors, the spaces for which are partially closed. On the right side, the first permanent molars are biting end-to-end; on the left, there is a

Fig. 15.—Profile of patient whose casts are shown in Figs. 13 and 14. A slight double protrusion and a short mandible. Possibly also a distoclusion case.

so-called distoclusion. The occlusal view of these casts is shown in Figure 17, where the transverse lines indicate that there is slightly more forward translation of the maxillary teeth on the right side than on the left. On the mandibular cast, this line shows that there is definitely more forward translation on the right side than on the left, which accounts for the end-to-end occlusion on the right side. The arches are somewhat under normal width, and a Hawley chart placed over the maxillary cast showed that the expansion in that arch was required mostly on the left side. The left mandibular lateral incisor erupted labially, whereas the right one erupted lingually and permitted the deciduous cuspid to pass it and drift forward, followed by all those posterior to it, and forcing the incisors somewhat to the left. Had this forward drift not taken place on the right side of the mandible, there would have resulted a so-called bilateral distoclusion, if we consider only the teeth, but Figure 18 shows a pretty satisfactory mandible and a fairly well-balanced face. The expansion of the dental arches to normal width would not

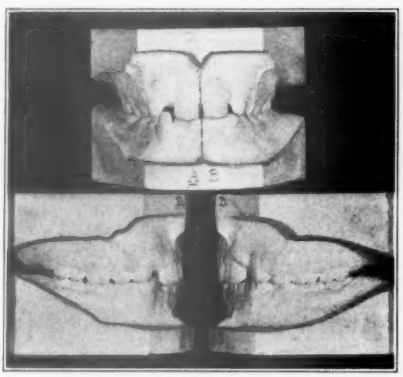

Fig. 16.—A mixed denture of a boy nine years of age. Maxillary lateral incisors not yet erupted, and the spaces partially closed. Axial relations not normal.

supply sufficient space for the maxillary lateral incisors without carrying the central incisors forward, and to bring the mandibular teeth on the left side forward to match those on the right side would make it necessary to carry all incisors farther forward, thus producing a double protrusion. In other words, all the teeth would be forward of their normal relation to the apical base and to the cranium. This is a neutroclusion case, and the malocclusion has been brought about by forward translation of the teeth in varying

degrees in different sections of the dental arches.

TREATMENT

What is the procedure in treatment? As the boy was nine years of age, and well developed, the first deciduous molars should soon be shed. Lingual wires were placed in October 1931, in order to bring about normal arch width, additional resistance being obtained on the right side of the maxillary arch in order to gain more expansion on the left side than on the right, as indicated. In the mandibular arch, the expansion required was more nearly equal. About eight months later, all four first deciduous molars were removed to facilitate eruption of the first bicuspids. After another four months, the mandibular left second deciduous molar was removed, because of pulp involvement. The lingual wires were removed in June 1933, one year and eight months after the original placement. The remaining three second deciduous molars were removed in October of that year. In June of the following year, study casts were made. The occlusal surfaces of these were shown earlier in Figure 4. The arches had narrowed a little from the width that had been obtained. Some time previous to this, the decision had been made to remove the four first bicuspids as soon as they erupted, as all the teeth were very large, and roentgenograms showed the presence of all the third molars.

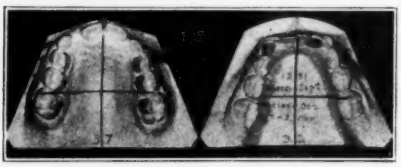

Fig. 17.—Occlusal view of casts shown in Fig. 16. Greater forward displacement on right side than on left.

In June 1934, the first bicuspids were removed and the pin appliance placed on both arches. Bands were fitted to all teeth sufficiently erupted. It was not yet possible to place bands on the mandibular second bicuspids, but these were a little later attached to the arch wire. U springs, with half-round pins attached, were placed to intrude the mandibular cuspids and carry the right one distally. As soon as the maxillary cuspids were sufficiently erupted, U springs were fitted to rest against the mesial surface of these teeth and start their distal movement. Later, these teeth were banded and springs with pins applied to carry them distally a little —roots and crowns, the one on the right more than that on the left. From the median lines on the casts (Fig. 4), it is evident that both maxillary and mandibular incisors had to be moved to the right. When the cuspids were in place, these teeth were attached to the arch wire and the springs adjusted to the lateral incisors, and finally to the central incisors. Nearly all incisors required some intrusion, particularly those in the mandible, in order to establish a normal overbite, 1.5 mm.

Fig. 18.—Profile of boy whose casts are shown in Figs. 16 and 17. This is a neutroclusion case.

Posterior teeth will move forward somewhat, when used as anchorage; but, in this instance, this was permissible, except in the case of the mandibular right first molar. Even in this case, where there was no maxillary protrusion, occipital anchorage was used somewhat. However, in many cases, where there is decided forward displacement of the maxillary teeth, occipital anchorage is indispensable; and, in older patients, in whom the second molars are sufficiently erupted, these teeth are also utilized as anchorage. For some time, I have been placing two vertical tubes on the first molars, and pins on the arch wires for these, which absolutely prevents tipping of these teeth and thus increases the anchorage very much.

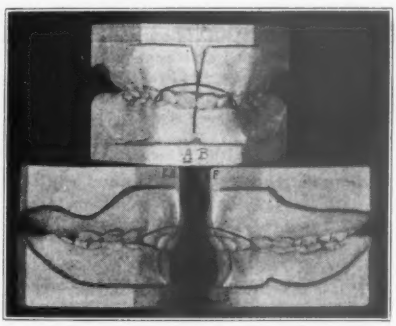

Fig. 19.—A mixed denture, showing excessive downward growth of premaxillae. Permanent central incisors were not allowed to erupt beyond normal plane of occlusion. (See text.)

Considerable time has elapsed since appliances were first placed in this case, but all that was done in the early treatment with the lingual wires was to gain expansion and retain the spaces of the deciduous molars until their successors erupted. It will be noted also that there was one year during this period when there were no appliances on at all. The actual correction of the case began about two and a half years ago. It is now almost completed and will require no retention appliances of any kind.

If no teeth are removed, the technic is somewhat different. It is described in several of my former papers. One of these cases, the casts of which are shown in Figure 19, was presented in a paper[8] before the Section on Orthodontia of the A.D.A., and has an added feature of interest. There was an excessive downward growth of the premaxilla carrying the deciduous incisors. There was also some over-eruption of the mandibular permanent central incisors, the resulting overbite being 7.5 mm. When the permanent maxillary incisors began to erupt, I stopped their downward progress when a little less than 3 mm. of their crowns were exposed. Figure 20 shows roentgenograms of the maxillary incisors at the time the appliance was adjusted to prevent their further downward movement, and later when their roots were fully formed.

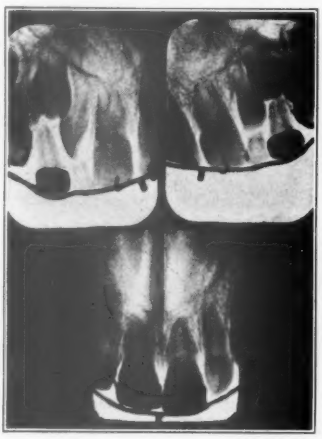

Fig. 20.—Same as Fig. 19. Above, at time arch-wire was placed to prevent further eruption of incisors; below, after completion of root formation.

These pictures show that the roots of the maxillary incisors went on to normal development regardless of the restraint placed on their eruption, and the excessive downward growth of the maxillary alveolar process was absorbed, exposing the crowns of the incisors normally, just

as would happen where teeth were intruded. This case was not quite completed to my satisfaction before the little girl went abroad to attend school, but the appliances were removed, and casts made. (Fig. 21.) The maxillary lateral incisors were under normal size, particularly the left one; which, along with a few other details, accounts for a little space between the maxillary right cuspid and first bicuspid, and also between the left maxillary incisors.

CONCLUSION

It is now generally conceded that growth of the jaws takes place occipitally.

The researches of Leuman M. Waugh

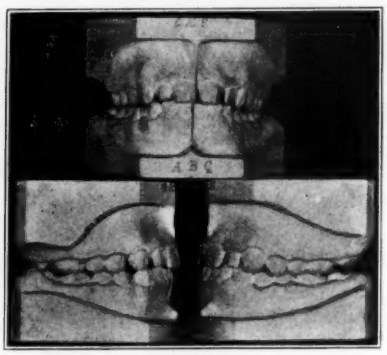

Fig. 21.—Later casts of case shown in Figs. 19 and 20.

and Weston A. Price demonstrate that where there is sufficient function of the dental apparatus, there is normal growth and development of the jaws and absence of malocclusion of the teeth.

Students of biology, as well as experienced clinical operators, agree that teeth drift forward of their normal relation to the apical base, owing to lack of normal backward growth of the jaws.

Therefore, the natural explanation of the anomaly of malocclusion is that, in all except the more simple cases, it is the result of forward translation of the teeth.

The function of the orthodontist, then, in treatment of these anomalies, must be the correction of any forward translation of teeth which has taken place, and the proper application of stimulus to bring about normal growth of the jaws where this is lacking.

Finally, where the forward displacement cannot be fully corrected without expanding the dental arches beyond what is normal, some teeth must be sacrificed, as it is not possible to carry back the crowns of all the teeth in both dental arches beyond the axial positions of the apical ends of their roots. Neither would it be practicable to do so, as impaction of many third molars would result.

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DIFFUSE FIBROMA OF THE GUMS

REPORT OF TWO CASES

By HOWARD J. BUCHNER, D.D.S., Oak Park, Ill.

THERE is a massive hyperplasia of the gingivae in young people which is rather rare. This condition is characterized by a luxuriant increase in the subepithelial connective tissue over an unchanged alveolar base distributed symmetrically about all or nearly all the teeth. The etiology is obscure and no common factor such as local irritation can be demonstrated. Treatment, other than radical excision and in some instances extraction of all the teeth, is wholly ineffective. In many cases, the occlusion is highly defective, and as a result of the malocclusion and the excessive amount of tissue, there is likely to be a marked facial deformity.

This condition has been described under a variety of names, the terminology being dependent apparently partly on the theory favored by the particular writer with reference to the genesis of the condition and partly on its resemblance to overgrowth in the other parts of the body. Some of its designations are: hypertrophic gingivitis, chronic hypertrophic gingivitis, hypertrophy of the gums, diffuse symmetrical fibroma of the gums and elephantiasis of the gums. The first two favor the etiologic factor of chronic irritation, the next two relate the condition to some disorder of growth and development and the last is based on a fancied resemblance to tropical elephantiasis. Weski[1] concludes, on the basis of the study of several cases, that this condition is of hereditary origin. Colyer[2] states that "such abnormalities as molluscum fibrosum, defective mentality and massive development of the hair are often present in cases of enlarged gum tissue."

Two cases have been carefully studied with the hope of throwing some additional light on the problems concerned with the etiology and treatment of this condition.

CASE 1.—*History.*—A school girl, aged fourteen years and four months, who came to the Orthodontia Clinic of the Northwestern University Dental School for treatment, had a vast hypertrophy of gum tissue throughout the mouth. The presence of hypertrophied gum tissue had been observed by the parents when the patient was three years of age. Information was lacking as to whether this condition existed prior to that time. The tonsils and adenoids were removed at the age of twelve. Previous to that time, the patient breathed through the mouth. Other than the fact that the mother and brother had the same condition of the gums, but to a lesser degree, the rest of the history revealed nothing which seemed to be abnormal.

Clinical Examination of the Mouth.—The teeth were in Class I malocclusion (Angle) with marked labioversion and separation of the upper anterior teeth. The saliva was very thick and viscous. The soft tissue was much enlarged, with only one-third of the crown of each tooth exposed. The tissue was firm, characteristically stippled and blanched, and tough and fibrous, with no apparent inflammation. On the labial and buccal surfaces of the upper arch, the tissue extended

(Read before the Section on Therapeutics, Pathology and Research at the Seventy-Third Annual Midwinter Clinic of the Chicago Dental Society, February 17, 1937.)

1. WESKI: *Deutsche Monatsschr. f. Zahnheilk.*, 38:557-584, 1920.

2. COLYER, FRANK: Dental Surgery and Pathology. New York: Longmans Green and Company, 1931, pp. 450-455.

approximately five-eighths of the distance from the crowns of the teeth to the reflection of the lips and cheeks. On the lingual surface in the tuberosity region were folds of movable tissue about 8 mm. thick. The soft tissue of the lower arch was not so extensively enlarged and not so characteristic as that of the upper arch.

Roentgenographic examination showed that the teeth were fully erupted, with the bone normal in appearance.

Systemic Examination.—After making a complete physical examination, Dr. Rony of

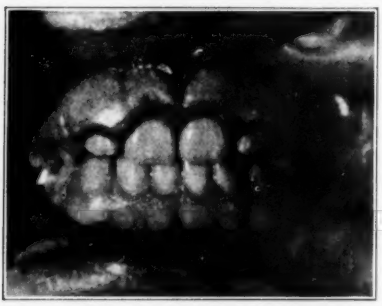

Fig. 1.—Case 1 before surgical treatment.

Fig. 2.—Case 1, front view, after resection.

the Northwestern University Medical School made the following statement: "This case represents a type frequently found in young girls in the period of puberty. There is a certain maladjustment of the endocrine and nervous system present." (There were no other defects and no excessive hair formation.) "The prognosis is good. Usually, no medical attention is necessary, adjustment taking place in time, automatically. Administration of whole ovary by mouth or some female sex hormone preparation hypodermically may be indicated." Whether the enlargement of the gums has any relation to this condition remains to be seen."

Treatment.—(a) Orthodontic treatment: When the patient applied for orthodontic treatment, appliances were placed. Correction of the teeth was carried on for one year. At this time, the appliances were removed, and an attempt was made to treat the hypertrophied gum tissue, which had been aggravated somewhat as a result of the orthodontic treatment.

Fig. 3.—Case 1, right side, after resection.

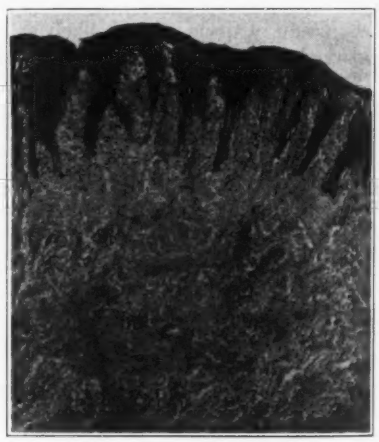

Fig. 4.—Histologic section of tissue from Case 1.

(b) Radium treatment: The upper arch was exposed to six 12.5 mg. platinum tubes of radium placed approximately 3 mm. from the tissue. Six exposures were made as follows:

6-29-31	½ hour	7-27-31	¾ hour

8-24-31	1½ hours	11-5-31	4 hours
10-5-31	2 hours	12-10-31	4 hours

Results. Immediately after exposure, the tissue appeared definitely inflamed. However, the tissue did not decrease in size. (Figure 1.)

(c) Surgical treatment: Since the radium therapy did not have a desirable effect, an attempt was made to excise the redundant tissue. On the labial and buccal sides, all the tissue was removed to the periosteum from the free gingival border well beyond the margin of the abnormal tissue. On the lingual aspect, anteriorly to the second bicuspids, the tissue was removed to the level of the bone, as is done in surgical treatment of pyorrhea. From the second bicuspids distally, the large fold of tissue was completely removed to the level of the periosteum. The areas surgically exposed were protected with

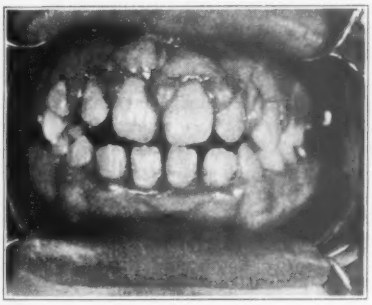

Fig. 5.—Case 2 prior to resection.

a layer of zinc oxide and eugenol paste. A solution of 8 per cent zinc chloride was applied to the inflamed areas for its astringent and cauterizing effect.

The new tissue formed after healing had taken place was a normal healthy-appearing gum tissue. Four years have elapsed since the surgical work was completed, and there is no recurrence of the hypertrophied tissue. (Figures 2-3.)

Histologic Description.—The cuticular layer is thickened. The epithelium is much wider than is usually found. The papillary extensions of the epithelium are long, and, in most areas, are definitely outlined. The connective tissue is very dense and coarse. (Fig. 4.)

Case 2.—*History.*—A girl, aged fourteen years and seven months, who came to the Orthodontia Clinic of Northwestern University Dental School for treatment, had, in the previous case, hypertrophied gum tissue. This condition was observed by the parents at the time of the eruption of the permanent teeth. The tonsils and adenoids had been removed at the age of three years. The patient had always been a mouth-breather. A minor resection of the hypertrophied gum tissue had been performed in October of 1929, before the case came under my observation. The growth recurred as much as before on the upper jaw, but to a lesser degree on the lower. There was nothing else of interest revealed in the case history.

Clinical Examination of the Mouth.—This

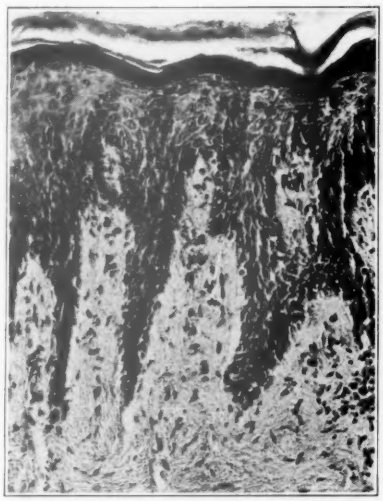

Fig. 6.—High magnification of surface tissue in Case 2.

description is made of the mouth as it appeared in the Fall of 1930. Although some orthodontic and surgical work had been performed, the condition was essentially the same as originally. The teeth were in Class II Division 1 malocclusion (Angle). The anterior teeth were not only in labioversion, but were also extensively rotated and in infraversion. The saliva was very copious and thick and stringy. The interseptal and gingival tissue was very much enlarged and inflamed, covering about two-thirds of the crowns of the upper teeth. There was no sharp line of demarcation between the ab-

normal and the normal areas. The tissue was not so tough and fibrous as that in the previous case. The enlargement was confined to the gingival region and was characterized by the presence of inflammation, which seemed to be aggravated by an irritation.

Systemic examination: A report from the medical school stated that the findings substantiated the conclusion that the patient was normal in every respect, with no other deformities and no excessive hair formation.

Treatment.—(a) Orthodontic treatment: As in the previous case, since the patient applied for orthodontic treatment, corrective measures were carried on for about two years. In the meantime, an attempt had been made to remove the hypertrophied tissue, only to have it return in excess on the upper jaw and to a lesser degree on the lower.

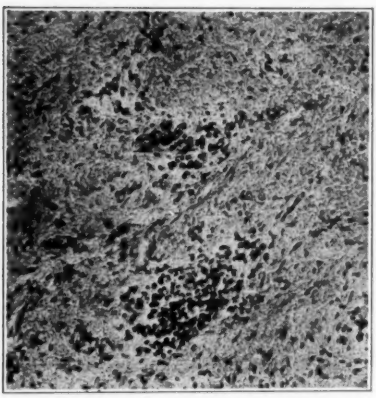

Fig. 7.—High magnification of tissue in Case 2 at deeper level.

(b) Radium treatment: Radium treatments were given on the upper and lower jaws as follows, the center column referring to the upper jaw, the right column to the lower:

5-25-31	½ hour	½ hour
6-29-31	¾ hour	¾ hour
7-27-31	1½ hours	1½ hours
8-24-31	1½ hours	1½ hours
10-5-31	3½ hours	3½ hours
11-3-31	3½ hours	3½ hours
12-1-31	3¼ hours	2¾ hours

Results. Inflammatory changes immediately followed the treatment, as in the previous case. When the time exposure was increased to three and a half hours, soreness was present in the area of tissue covered by the tubes one week after exposure and lasted until the end of the third week. After six months of treatment, the tissue in the area of the tubes was white and scorched in appearance. The tissue was not decreased in size. (Fig. 5.)

Surgical treatment: As stated above, resection had been carried out with no success on the upper arch. However, since the first case responded so nicely to surgical measures, it was thought that perhaps this case would respond more favorably if all the redundant tissue were removed and extreme care taken to prevent the formation of inflammatory tissue during healing. In the summer of 1933, complete surgical removal of the hypertrophied tissue was carried out in the same manner as in the first case.

Three months after resection, a slight recurrence was observed in the upper right bicuspid region. This area was resected again in January 1934. Since that time, there has been a very slight recurrence.

Histologic Description.—The cuticular layer is thickened. The epithelium is much wider than is usually found. The papillary extensions of the epithelium are very long, and in most areas are definitely outlined. Occasionally, the outline is less well defined because of leukocytic infiltration at the base of the epithelium extending a short distance into the connective tissue, which is very dense and coarse. In the deeper layers, there is quite a noticeable leukocytic infiltration surrounding the blood vessels. (Figs. 6 and 7.)

COMMENT

1. Conditions similar to those herein presented have been described as enlargement of the bone tissue. In the tissue resected, the bone tissue appears to be normal in dimension, the enlargement being wholly in the gum tissue.

2. The tissue possesses tendencies to react to irritation more readily than normal tissue.

3. The first case seems to be hereditary, since the mother and brother have a similar condition.

4. Both cases were accompanied by

extreme malocclusion and characteristic saliva, but presented no other deformities and no excessive growth of body hair.

5. Such tissue does not respond to radium in the dosage used.

6. Surgical treatment was successful in the first case and partially successful in the second.

7. It is necessary to remove the diseased tissue completely, and prevent the early formation of inflammatory tissue.

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REMOVAL OF IMPACTED TEETH

By James A. Blue, D.D.S., F.A.C.D., Birmingham, Ala.

IMPACTED teeth wherever found should be roentgenographed from different angles, by the operator, or some one familiar with his technic, to determine their relationship with other teeth, bone interference, curvature or enlargement of roots and proximity to major nerves and blood vessels. In discussing the maxillary third molar, it will be observed that there are no large vessels or nerves to contend with in its removal; but we must keep in mind the relationship of the third molar to the distal portion of the floor of the antral cavity.

The x-rays are a very valuable aid in locating this tooth, provided two or three views are made, one view in the usual manner showing the crowns and roots of the first and second molars. Next, a view is made by bringing the tube nearer the front of the face, directing the rays distally and at the same time raising the tube higher than in the first view. This will determine whether the impaction has a buccal or palatal inclination. The third view, with the tube moved distally even with and above the ear, directing the rays forward and downward, will give a fair conception of the root formation.

The average maxillary third molar has only a soft tissue interference. The technic for the removal of such a tooth is as follows: An incision is made beginning midway between the distobuccal and the distopalatal angles of the second molar and extending distally into the mucous fold as far as possible. The mucoperiosteum is then stripped from the bone buccally, the tooth being thus exposed. Then the tissues are elevated toward the palate, more buccal than palatal elevation being necessary. A good sense of touch and proper instruments are all that is usually needed for removal of the average unerupted maxillary third molar.

For the deeply impacted teeth, the procedure is the same as just described, with extension of the buccal flap forward, elevating the mucoperiosteum as far forward as the buccal surface of the first molar. This flap is retracted to the buccal fold and affords a full view of the tuberosity. The overlying bone is removed, together with enough of the buccal bone to expose the coronal portion of the tooth, care being taken at all times to protect the roots of the second molar. An elevator applied to the palatal side, forming a wedge, will loosen the tooth and it may be removed buccally.

The impacted cuspid should be roentgenographed through the bicuspids and also through the central and lateral incisors. The third view should be made through the palate, the operator aiming at the bridge of the nose and directing

(Read before the Section on Oral Surgery, Radiology and Anesthesia at the Seventy-Third Annual Midwinter Clinic of the Chicago Dental Society, February 17, 1937.)

the rays down through the cuspid region. This will suffice in the average case and will determine whether the cuspid is on the palatal or the labial side of the adjacent teeth. If the tooth is on the palatal side, the operation consists of an incision extending from the median line along the gingival border, posteriorly to the mesial surface of the first molar. Then an incision is made in the median line extending back to the center of the palate; the flap is turned back, and the bone over the tooth is exposed to afford easy access to the operative field. A liberal amount of bone should be removed, the coronal portion and at least two-thirds of the root being exposed. The tooth is cut through at the neck with chisel and mallet and each section removed. The flap is replaced and securely sutured in position. A small ribbon of gauze is slipped through the center incision and into the tooth socket to allow the escape of excess blood, which always fills these sockets.

The mandibular third molar should be roentgenographed from at least three angles. First, the anterior border of the film is placed slightly distally to the mesial surface of the first molar, with the upper border one-fourth inch above the first and second molars and the rays directed between the first and second molars. If the third molar overlaps the second, it will appear to extend into the second molar. Another view is taken with the tube forward and the rays directed distally. If the third molar is overlapping buccally, it will appear in this view to have moved away from the second molar; if it is overlapping lingually, the overlapping will be more pronounced. For the third view, the tube is moved distally, and the rays are projected forward. Some information is thus obtained regarding the formation of the roots and their relation to the mandibular canal.

Flaps for impacted or unerupted mandibular third molars should begin midway between the distobuccal and the distolingual angles of the second molar, extending distobuccally and following the curvature of the bone in this region to the ascending ramus, and thereby, avoiding disturbance of the lingual tissues. The gingival tissues are elevated from the buccal surface of the second molar to the mesiobuccal angle. This procedure affords sufficient access to the bone on the buccal side, allows the flap to be retracted without undue strain and prevents tearing and laceration. The tissues along the lingual border of the bone are carefully elevated, extending forward sufficiently that the distolingual angle of the second molar is exposed. The necessary bone is removed and the tooth is elevated from its socket; or the tooth may be split with a chisel and removed in sections. This procedure often conserves bone and shortens the time of operation. The bone should be inspected for rough or sharp margins, and these made smooth. The flaps are then apposed and sutured, one suture usually being sufficient.

The use of a flap of the foregoing design, if properly handled, prevents excessive trauma, as the lingual tissues have not been cut or unduly lacerated. The buccal side is protected by the buccal flap and thus an opening at the distobuccal angle of the second molar, which so often occurs if incisions are made too near the distobuccal angle of this molar, is prevented.

Comer Building.

SHOULD IMPACTED TEETH BE REMOVED?

By A. Porter S. Sweet,* D.D.S., Hornell, N. Y.

SINCE the "Year Book of Dentistry" is supposed to contain the best thought in dentistry from all over the world and, since The Journal supplies about a third of its material, it would seem that a survey of The Journal for the last five years would give an answer to this question in line with the best thought in the profession.

Let us permit the authors to speak for themselves:

Horace M. Davis, in 1932, said:

Can any one here give a good reason why a useless tooth should remain in the mouth until it gives pain and forces the patient to have it removed, unless, by the removal of the tooth, a worse condition may be brought about? . . . Irregularities and deformities in the mouths of many children are caused by these teeth, as well as cysts, nervous diseases, etc. At a later period, it may be at 16 to 25 or later, we see, in addition to the foregoing, crowding of the anterior teeth and infected areas on the distal surface of partially erupted third molars. . . .Later in life, whether it be at 30 or 70, many of the diseases to which we are subject come under the heading of conditions in which the teeth must be considered as factors. To find at this stage of life that impacted teeth are present and must be removed under adverse conditions is an indictment of our previous attention to these patients. (Journal A.D.A., May 1932.)

Carl D. Lucas, in 1935, believed that:

Since impacted and unerupted teeth are organs which are devoid of physiologic function, they are identical to foreign bodies in the tissues. Normal tissues do not tolerate foreign bodies without reaction, characterized by inflammation of a type which promotes their exfoliation. Since unerupted teeth are not exfoliated, their supporting tissues are chronically inflamed; hence, the sites of such malposed teeth are foci of deranged physiologic function. (Journal A.D.A., February 1935.)

The author further states that for the foregoing reason, these teeth afford an ideal place for infection to locate.

J. P. Wahl, in 1936, wrote:

It is my opinion that all impacted and malposed teeth, with a few exceptions, should either be placed in their normal position or be removed. . . . I have made it a routine practice before operating to determine whether there was any communication between the completely impacted or malposed tooth and the oral cavity. In 75 per cent of the cases examined, I found an opening. . . . In the presence of malposed or impacted teeth, as the patient becomes older, he may be affected with reflex pains, epilepsy, chorea, facial spasms, dementia precox, mania, paralysis, sensory alterations, neuralgia, paroxysmal pains, insomnia, muscular twitching, melancholia, neuritis, arthritis and loss of memory. Some cases of loss of vision have been reported. (Journal A.D.A., July 1936.)

D. J. Aubertine, in 1936, said:

The dental profession has been too prone to allow the impacted third molar to remain if the patient is not complaining of any untoward symptom at the time. . . . If it has been definitely decided that it would be impossible for these teeth to erupt normally in the arch, it is better to remove them at this time, rather than later in life when the roots are fully formed. (Journal A.D.A., December 1936.)

This author then continues with the observation that in every case of tic douloureux, in his experience, there had been a history of either impacted teeth or pulp stones.

It seems to me that these authors have answered our question in a very lucid and satisfactory manner. Since these teeth are such potential "powder-kegs" of trouble, why should we temporize and procrastinate? Impacted teeth should be removed.

(Read at a meeting of the Rochester Dental Society, April 19, 1937.)

*Staff dentist, St. James Mercy Hospital.

THE JOURNAL *of the*
AMERICAN DENTAL ASSOCIATION
and THE DENTAL COSMOS*

C. N. JOHNSON, Editor

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*In deference to the wish of the dental profession to control its literature, *The Dental Cosmos,* founded in 1859, was transferred by the publishers as a gift to the American Dental Association, January 1937.

Editorial Department

THE CHRISTMAS SEALS

IT is customary to remark that one Christmas is much like another, but this is the remotest thing possible from the truth.

No two Christmases are ever the same or ever can be the same. The shades of difference may escape the ordinary observer, but they are there if only we had the vision to recognize them. And what a blessing this is! Imagine the drab and dreary world it would be if every experience of life were merely a duplication. Even if it were possible for us to repeat experiences again and again, which it is not, the result would ultimately cloy our senses and leave us stale and uninterested.

The spice of life is achieved only by the bombardment of new ideas, and the impact of new experiences. Most of us would perish of *ennui* if we were forced too rigidly into routine, and thus it is that Christmas would mean little to any of us if every Christmas were precisely the same as every other Christmas.

Of course, there are distinguishing characteristics in every Christmas

whereby the highlights may be recognized. Whenever we think of Christmas, we think of sleighbells—some of us do. We think of tinsel and glitter and bright lights. We think of homecoming and of ruddy faces; of the Yule log, and of the mistletoe and holly; of roast turkey and cranberry sauce; of Christmas trees by the hearthstone, and even out on the lawn, with wondrous colored decorations lighted up in modern times with myriad-hued brilliance. We think of the blessed legend of Saint Nicholas and the clatter of hoofs on the house top. We think of distant chimes floating over the hills and across the valleys—all warming our hearts with their memories. But, most of all, we think of the prattle of little children, and the small shrill cries of delight, and the patter of little feet running from room to room, and the eager chubby hands reaching up in tremulous glee to hang the stockings by the chimney corner.

After all, Christmas is the heyday of the blessed children, when the wee ones of earth come nearer to the human heart than at any other time, when frivolity is not folly, and the seriousness of life is wisely pitched in a minor key. It is here that the beneficent blend of sanctity and hilarity is at its height, and the souls of men expand without the bane of pride or vaingloriousness.

Christmas is the time when the days are short and the nights are long, when the fireside means more than at any other time, when the blessings of warmth and light enter the hearts of humanity and make the whole world kin, when rich and poor, and fortunate and unfortunate—ah yes, alas, there are always the unfortunate, and at this season they must not be forgotten, when we must make common cause with those who are afflicted and those who are in need. Christmas comes as a blessed benefaction to those in our own profession who have fallen on evil days and felt the pinch of adversity. It is always sad to think what the plight of many in our ranks might be were it not for the vision that came to a few of the noble souls in our Association who first conceived the idea of bringing relief to the needy among our members, and started the Relief Fund.

Each Christmas brings with it the cherished privilege of sharing in the beneficence of this truly worthy and wonderful project that has brought comfort and happiness to the homes of many who would otherwise have been in need. The Christmas seals have proved an appropriate and happy medium of annually augmenting the fund to maintain this philanthropy, and the members of the Relief forces have administered the fund in a most efficient and praiseworthy manner.

Members of the profession need have no hesitancy in subscribing one

hundred per cent for the seals. In fact, it should be considered not only a duty, but also a pleasure to do this. The depression of recent years in some instances made it a real sacrifice to spare the dollar, but now that our economic situation has happily improved, the response from the seals should be correspondingly augmented.

This Christmas let us expand our hearts and sanctify our souls by a generous display of helpfulness and benevolence that shall gladden the homes of every one of our less fortunate brother dentists. Our entire membership will be benefited, the ones who receive and, more than all else, the ones who give.

ARMY DENTAL CORPS LEGISLATION

At the seventy-fifth session of Congress, the House of Representatives, on the recommendation of the Committee on Military Affairs, passed a bill authorizing an additional number of medical and dental officers for the Army. The bill is now on the Senate calendar as Senate Bill 2463 and is expected to come before the present special ssesion for action by the Senate.

This measure, presented in the House of Representatives by Congressman Collins of Mississippi, was strongly championed by Mr. Harter, Congressman from Ohio, who, as a member of the Military Affairs Committee, referred the bill to the Senate. In referring the bill to the Senate, Mr. Harter makes an eloquent appeal for the passage of the bill by the Senate based upon a most comprehensive and sympathetic understanding of the desirability of an increase in the Army Dental Corps to meet in a somewhat satisfactory manner the urgent need for more extended dental service in a constantly increasing Army.

On the record of the Dental Corps since its development from the humble status of a contract dental service to its present commissioned status, it would seem that no argument should be necessary to justify the increase in available dental service called for by the normal increase in the Army personnel. When the Dental Corps was first established in 1911, it was on the basis of one dental surgeon to every one thousand enlisted men in the Army, and it was even then recognized that this was inadequate for the needs. Notwithstanding that, the Dental Corps has valiantly

struggled against the odds throughout the intervening years until today the task has become so great that continuation of the present inadequacy in physical equipment in this branch of the health service might actually threaten the survival of this important branch of the Government service.

The increased demands upon the service of the Dental Corps during the World War resulted in a very substantial increase in the Corps during the war, but, some time thereafter, the proportion was again reduced to the number specified in the original legislation establishing the Corps in 1911; so that now for some twenty-six years the Army Corps has been on the basis of one dental surgeon to every one thousand enlisted men—and more.

Every one will readily admit that this is a distressingly inadequate provision for the ordinary dental service provided by the Corps, to say nothing of the demands created by the continuously enlarging scope of the field of the dental surgeon. This is particularly apparent to those who are cognizant of the scope of service of the Corps, and Mr. Harter's comments on the state of the Corps, which are based on his official contacts, are or should be convincing beyond doubt as to the desirability and necessity of a substantial increase in the Dental Corps.

It is not as though we were endeavoring to prove the value of dental service as a health measure in the Army—we have long since proved that, under the pressure of actual war conditions and, too, in what may be termed an apprenticeship of some ten years as contract dental surgeons previous to the establishment of the commissioned Corps.

In that trial period, a group of pioneers, with practically no social or military status, undertook the task of demonstrating the value of dental service to the Army. How well they succeeded is today a matter of history. And from that early effort, the dental corps of the army has developed an efficiency of service as a health asset to the Army that has elicited the praise and admiration of the entire military service.

The possibilities of the benefits of dental surgery in reducing the percentage of disabilities arising from dental and oral conditions have been made amply evident, and it has also been demonstrated that the particular oral diseases which the dental surgeon is called upon to treat can best be cared for by those who are trained and skilled in dental surgical technic. And then again, during the World War, the dental surgeon once and for all disposed of that ancient fetish that diseases and lesions of the mouth and jaws can be properly cared for only by those who have had the training and instruction furnished by the medical curriculum. They demon-

strated the possibilities of and thereby created a need for specialized training in connection with the lesions which affect the dental and oral tissues, and paved the way for world-wide recognition of the importance of dental health service in the military arm of the Government.

The record here sketchily recorded seems to us to fully justify not only the request from the profession for the increase in the number of dental surgeons in the Corps, but also the higher social and official status that would be accorded to the Dental Corps through conferring upon its head the title of Brigadier General.

It is anticipated that Senate Bill 2463 will come before the special session for action, and with that thought in mind the Chairman and Legislative Committee of the American Dental Association urgently request that each member in his own way use his most effective influence directly or indirectly upon the senatorial representative from his particular district, to the end that the Army Dental Corps may be placed upon a basis of efficiency worthy of the military branch of probably the strongest exponent of peace in all the nations of the world.

It has become axiomatic that the greater the preparedness for war, the greater is the probability of peace. And while we disclaim any desire for belligerency now or in the future, we do believe that preparedness in the form of health service for the Army is a worthwhile and distinct peace asset.

L. P. A.

DENTAL RELIEF FUND COMMITTEE

THE Relief Fund Committee for the State of North Carolina reports, under date of November 11, that the Raleigh Dental Society of the North Carolina Dental Society has already contributed 100 per cent to the Relief Fund.

This is an example of promptitude and generosity well worthy of emulation by every District and State Society in the American Dental Association.

The Dental Relief Committee is anticipatively hopeful that this highly encouraging note portends the nature of the general response to its efforts for the coming year.

FRED R. ADAMS, *Chairman*,
Dental Relief Fund Committee,
55 West 42d Street, New York City.

CORRESPONDENCE

WE take pleasure in reproducing, herewith, a letter to the President of the American Dental Association, from the Chairman of the Social Security Board, Mr. A. J. Altmeyer. The letter is self-explanatory.—EDITOR.

November 2, 1937

Dr. C. Willard Camalier,
President,
American Dental Association,
1726 I Street, N. W.,
Washington, D. C.

Dear Dr. Camalier:

I have just read the editorial in the October issue of THE JOURNAL OF THE AMERICAN DENTAL ASSOCIATION dealing with the paper "Building the Social Security Program," which I read at your Atlantic City sessions in July. I regret very much if the paper was printed in another periodical before you had an opportunity to consider whether you wanted to publish it in your own Journal. I had no knowledge that you intended to print it or that any other organization would print it. In making the paper public on the day it was delivered the usual governmental procedure was followed of giving out public material to the press on a subject of public interest.

What concerns me more, however, is the fact that I did not make myself clear with regard to the problem of "health insurance." I tried to point out in my paper that the problem of health insurance should be broken down into two elements, namely, medical care and benefits from wage loss. This separation of the two items usually considered in any discussion of health insurance seems very important to me and I tried to indicate that in the Social Security Act we already have two plans for social insurance against loss of wages which might be extended so that protection could be offered against loss of income due to temporary and permanent disability. I tried to make it very clear that "the problem of providing *health services* would be approached in the light of the measures which have already been successful in our experience with public health and other public welfare services" but I made no suggestion whatsoever that the Social Security Act be extended to provide medical benefits under any type of insurance plan.

Students of both medical economics and social insurance have suggested to me that the *economic* risks of disability might be met by amending state unemployment compensation acts to include benefits for wage loss due to temporary disability and by amending the federal old age insurance plan to include benefits for wage loss due to permanent disability.

It seems to me that the payment of such benefits for wage loss might be of great advantage to both dentists and doctors since neither plan would encroach upon the rights of the professional man but rather make it possible for his patients to pay for more adequate professional service when needed.

I sincerely trust that we may have an opportunity soon to discuss this problem which I appreciate is of great concern to the dental profession. Please be assured that the Social Security Board welcomes your offer of cooperation to the end that a constructive solution may be found.

Sincerely yours,
A. J. ALTMEYER, *Chairman,*
Social Security Board,
Washington, D. C.

ASSOCIATION ACTIVITIES

LEGISLATIVE COMMITTEE

A matter of first importance to the dental profession is the pending Senate legislation that seeks to provide for additional officers for the Army Dental Corps and the rank of Brigadier General for the Chief of that service.

The Committee on Legislation calls on all members who are concerned with the standard of health service in the Army, the welfare and efficiency of the Army Dental Corps and a proper recognition of the importance of dentistry as a health service, to wire or write their Senators at Washington requesting support for S. 2463, a bill that must be adopted if the Army is to receive a minimum of adequate dental service.

A. D. PATTERSON, D.D.S., *Chairman,*
Legislative Committee,
Peoria, Ill.

DISCUSSION OF THE BIEMILLER BILLS PRESENTED AT THE LAST WISCONSIN LEGISLATURE

DURING the past session of the Wisconsin Legislature seven bills pertaining to socialization of medicine and dentistry were presented by Assemblyman Andrew J. Biemiller (Socialist), Milwaukee. The bills also had the support of some labor organizations. Their presentation was predicated upon the supposition that medical, dental and hospital care could be better dispensed and at much less cost to the individual if presented through these socialized plans. This thought is quickly absorbed by the citizens of a community who are naturally thinking of medical service primarily from a fee standpoint. They have become so accustomed to receiving a high type of medical service that they give no consideration to the fact that regimentation of the service might also jeopardize its quality.

As soon as the Biemiller bills were presented, the Wisconsin State Medical Society, the Wisconsin State Dental Society and the Wisconsin Hospital Association made a thorough study of each individual bill. They diagnosed them according to the values that would be received by the patients. Under the direction of Mr. J. G. Crownhart, secretary of the state medical society, a comprehensive résumé of each bill was made. In pamphlet form, this was presented to the individual physicians and dentists of Wisconsin and to other interested organizations. Group meetings were held of physicians and dentists in various parts of the state to hear Mr. Crownhart present a discussion of the bills. Several large newspapers, sensing the dangers of socialized medicine, gave editorial space condemning the bills. The individual members of the profession, now fully instructed as to the contents of each bill, made known to their patients the objectionable features of these bills. The response from the citizens of the state showed that they were not as yet willing to gamble away so valuable an adjunct of their daily lives as their present medical-dental services. The one fact that the presentation of the Biemiller bills established is that the people do value the high quality of the med-

ical, dental and hospital care which they receive in Wisconsin. There are plenty of socialized systems in Europe that can be presented in comparison with services rendered in this country. According to authorities who have personally studied the European systems, no one system has in any case bettered conditions for the patient or the profession.

The number of men in the profession is comparatively small, but the number of patients involved ultimately include practically the entire population. Jeopardizing the entity of our profession when considered relatively in numbers would not cause much of a disturbance, but jeopardizing the medical service to be received by the people of a country would be a calamity. The experience of the professions in Wisconsin emphasizes the necessity for the doctors to make more use of the literal translation of the term "doctor" and to really include "teaching" in their daily routine.

There are some who feel that the Biemiller bills would have been defeated without the coordinated opposition offered by the medical and dental societies. That would have been legislative. As it now stands, more people not connected with the professions see socialized medicine from the standpoint of value received rather than from the standpoint of fees. Several of the Biemiller bills died with the adjournment of the legislature. Those that were balloted on were defeated by a vote of approximately three to one. There will undoubtedly be further attempts to introduce legislation in Wisconsin such as the Biemiller bills. The people of Wisconsin, however, will be very slow in accepting regimentation of the professions to replace the present system.

The officers and legislative committees of the Wisconsin Medical and Dental Societies deserve much credit for their efficient opposition to these bills. They appeared at Madison at the several hearings on these bills and also contacted component society officers in each community for the dissemination of facts concerning the bills to the individual members of the profession.

The following is a brief résumé of the bills as presented:

Bill 661-A. This provided for an interim committee to study costs of medical care in Wisconsin and for this they sought an appropriation of three thousand dollars. This would only be approximately enough to publish a report, without expenses incurred in obtaining the information and therefore was opposed by the societies. They feared the study would only be an accumulation of pre-conceived ideas.

Bill 662-A. Group Hospitalization Insurance Plan. This provided for group hospitalization on an insurance basis for which there exist at the present time no actuarial rates. No rates were stated in the bill. The various sizes of the groups insured seemed to make the certainty of benefits, at the time they would be required, very dubious. The bill was opposed by the societies.

Bill 740-A. Medical-Dental Hospital Care of Indigent. This bill made it a duty of the county board of supervisors to see that the county cared for the medical and dental hospital needs of those receiving relief. It was opposed by the societies because it limited the agencies through which services to the needy could be rendered and made no provisions for border line cases which now receive consideration. The societies do favor the county system.

Bill 747-A. This bill would permit communities to subsidize or employ *tax-supported* physicians to care for all residents; under control and rules of municipalities or county board whenever there shall be an insufficient number of competent physicians in their respective communities. The bill was opposed by the societies on the ground that a sufficient number of physicians exist for all territories and also because the physician would be under the

control of a lay-governing body and consequently be under continual pressure therefrom.

Bill 748-A. A bill permitting counties to erect and operate joint county hospitals, employ staff and other help necessary and treat (even as a tax-supported service) all county residents therein. Wisconsin already has a law permitting the creation of county hospitals singly or jointly. This bill was presented to make it possible for the county to employ physicians and surgeons to treat all persons in or out of the hospital at tax expense. The societies opposed this and several other phases.

Bill 850-A. A bill authorizing third party associations, including cooperatives, to buy or furnish medical services for uniform prepaid premiums wholly exempt from insurance supervision. This was one of the most vicious of the Biemiller bills. It provided for cooperatives to furnish medical or hospital care, would have opened the hospitals to chiropractors, osteopaths and midwives, provided absolute freedom of the cooperatives from restrictions of Wisconsin insurance laws, provided severe penalties for any individual or group of individuals who might attempt or be claimed to be attempting to obstruct the development of the cooperative organization and made it impossible for the medical society to refuse admission into their society of any one belonging to a cooperative organization. The bill was opposed as being basically unsound.

Bill 852-A. Compulsory Sickness Insurance. The societies opposed this bill affording insufficient funds and bureaucratic and third party determination of service rendered by physicians to the sick.

O. H. Moen, D.D.S., M.S.D.,
Watertown, Wis.

MALPRACTICE: OSTEOMYELITIS FOLLOWING EXTRACTION OF TOOTH

The defendant, a dentist, extracted the plaintiff's lower right second molar. Four days later the patient began to experience pain and again consulted the dentist. Finding inflammation around the lower right third molar, the dentist, after a roentgenogram had been made of it, recommended its removal. After the extraction, the patient continued to have a "grinding pain" and consulted a physician, who discovered that the soft tissues near the tonsils were infected. A peritonsillar abscess was lanced and later a diagnosis of osteomyelitis was made. Attributing the osteomyelitis to the extraction of the third molar, the plaintiff sued the dentist. The jury returned a verdict for the patient, and the defendant brought exceptions to the Supreme Court of New Hampshire.

The dentist testified that, while the third molar was not decayed, there was an abscess between it and the site from which the second molar had been extracted. He extracted the third molar, he stated, to obtain better drainage of the abscess. The gums were inflamed and spongy, and the alveolar process was absorbed, leaving the tooth very loose, just holding by flesh. When a tooth is loose in this manner, he testified, the space between the tooth and the gum forms a pocket for infection and under such circumstances it is good dental practice to remove the tooth. The only expert called by the patient was the physician who treated him. This physician disclaimed any qualification as "an expert on dentistry" and did not know the degree of skill and knowledge possessed by the average dentist practicing in the community in which the defendant practiced. The witness testified, however, that there is a principle in surgery never to remove a sound organ when it is surrounded by infected tissues; that the removal of a

sound tooth from an infected area permits a chance for seepage and allows more space for the infection to get into the blood and to become localized in the bone; and that, in his opinion, the removal of the third molar resulted in the osteomyelitis. Four dentists testified on behalf of the defendant and each testified that proper practice required the removal of the tooth. None of these dentists stated that it was not good dental practice to extract the tooth under the conditions described by the patient.

A person, said the Supreme Court of New Hampshire, who holds himself out as practicing a particular profession is required only to possess the knowledge and to exercise the care and skill of the ordinary practitioner of that profession in the same or similar localities. The burden was on the plaintiff to prove the liability of the defendant, but the court could find nowhere in the record any evidence that the defendant did not possess the learning and exercise the skill and care of the average dentist in the community in which he practiced or that he had departed from the standard imposed on him by law. A dentist does not insure his patients against infection following the extraction of teeth. Since the patient failed to produce any evidence with respect to the negligence of the defendant, the dentist's motion for a directed verdict should have been granted.

The Supreme Court accordingly entered a judgment for the dentist.—April v. Peront (N. H.) 188 S. E. 373, *J.A.M.A.*, 109 : 1072, September 25, 1937.

AMERICAN DENTAL ASSOCIATION SPECIFICATION NO. 8 FOR DENTAL ZINC PHOSPHATE CEMENT*

FIRST REVISION (EFFECTIVE JULY 1, 1938)

By George C. Paffenbarger,† D.D.S., and John R. Beall,† B.S., Washington, D. C.

The formulation of satisfactory specifications is one of the principal activities of the cooperative research on dental materials jointly conducted by the National Bureau of Standards and the Research Commission of the American Dental Association. To date, nine specifications for various dental materials have been developed. These are for: (1) Amalgam Alloy; (2) Inlay Casting Investment; (3) Impression Compound; (4) Inlay Wax; (5) Inlay Casting Gold Alloys; (6) Dental Mercury; (7) Wrought Gold Wire; (8) Zinc Phosphate Cement, and (9) Silicate Cement.

REVISION OF ZINC PHOSPHATE CEMENT SPECIFICATION

Since the publication of the reports[1, 2, 3] on zinc phosphate cements, these materials have been greatly improved. This improvement, noted in the routine testing of the guaranteed cements for compliance with the specification, was so great that it became feasible to make the zinc phosphate cement specification more restrictive. As is customary, the proposed revision of the specification was submitted to both consumers and producers for their suggestions. The following revised specification was adopted (to be effective July 1, 1938) by the American Dental Association during the 1937 annual meeting.

*A Report to the Research Commission of the American Dental Association. Publication authorized by the executive Board of the Research Commission.

†Research associate of the American Dental Association at the National Bureau of Standards.

A. GENERAL SPECIFICATION.

This specification is for dental cements, the primary uses of which are:

A-1. To join or to seal dental appliances to oral structures or to other appliances.

A-2. To serve as a base or foundation for other filling material.

A-3. To serve as a temporary filling material.

B. TYPES.

Only one type of material is specified. The consistency of the mix which different uses require can be varied by the individual operator to suit his own needs.

C. MATERIAL.

C-1. These cements shall consist of a powder and a liquid, which, when mixed in the proper dental manner, will harden, or set, and give satisfactory dental service.

C-2. The powder and liquid shall be uniform and free from poisonous foreign materials.

C-3. Colors for the set cement shall be specified by the purchaser.

D. GENERAL REQUIREMENTS.

D-1. The liquid shall be free from cloudiness, precipitates, deposits or sediment.

D-2. Cements when spatulated in the proper manner shall not:

D-2a. Form lumps or granules.

D-2b. Evolve gas.

D-2c. Discolor tooth structures.

E. DETAIL REQUIREMENTS.

E-1. *Time of setting.* The time of setting shall not be less than four nor more than ten minutes.

E-2. *Ultimate compressive strength.* The ultimate compressive strength shall not be less than 840 kilograms per square centimeter (approximately 12,000 pounds per square inch) for specimens crushed seven days after mixing.

E-3. *Film thickness.* The film thickness shall not exceed 0.040 mm. (0.0016 in.).

E-4. *Solubility and disintegration.* The solubility and disintegration of the cement shall not exceed 0.30 per cent by weight during immersion for seven days in distilled water.

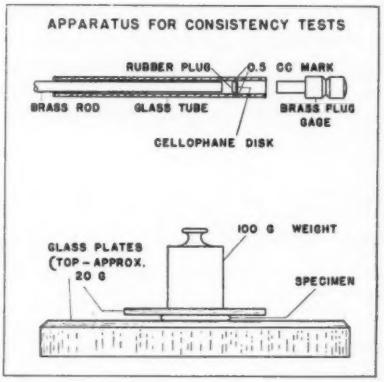
E-5. *Arsenic.* The mixed cement shall not contain more than one part of arsenic in 500,000 parts of cement (0.0002 per cent).

F. METHODS OF INSPECTION AND TEST.

F-1. *Preparation of test specimens.* The preparation of the test specimens shall be conducted at a temperature between 18 and 24° C. (65 and 75° F.) and at a relative humidity between 55 and 75 per cent. The powder-liquid ratio used shall be determined by the consistency test. The mixing technics employed in the preparation of all test specimens shall be those which accompany the packages of the cement, except that the temperature and humidity may vary within the above-mentioned limits.

All apparatus and instruments shall be clean, dry and free from particles of hardened cement.

F-2. *Testing consistency.* One type of apparatus for measuring consistency is shown in the accompanying illustration. This apparatus consists of a glass tube (inside diameter approximately 6.5 mm. [0.25 in.]), which will deliver a definite volume (0.5 cc.)* of mixed cement, two flat glass plates and a weight. The combined weight of the top plate and the weight shall be 120 gm. Trial amounts of powder shall be mixed with 0.5 cc. of liquid. Then 0.5 cc. of mixed but unset cement shall be placed in a measuring device and deposited on a flat glass plate. Three minutes after the mix is started, another glass plate (weighing approximately 20 gm.) and the additional weight shall be carefully placed on the soft cement. Trials shall be made until the average of the major and minor diameters of the slumped mass of cement shall be 30



*Unit actually employed is the milliliter (ml.), the one-thousandth part of a liter.

±1 mm. (1.18 inches) ten minutes after starting the mix. The average of the amounts of powder used in three such determinations shall be the amount of powder (combined with 0.5 cc. of liquid) necessary to produce a mix of standard consistency.

F-3. The *test for arsenic* shall conform to that designated by the American Chemical Society recommended specification for reagent phosphoric acid.[4] A detailed description of this test is given by Hillebrand and Lundell.[5]

G. TIME OF SETTING.

G-1. A ring 5 mm. (0.2 inch) high and 10 mm. (0.4 inch) in diameter shall be placed on a flat plate and filled with cement of standard consistency. Three minutes after starting the mix, the specimen shall be transferred to an atmosphere of 100 per cent relative humidity at 37° C. (99° F.). A standard Gillmore needle (weighing 454 gm. [1 pound] and having an end 1.06 mm. [0.0417 inch] diameter) shall be lowered vertically until the surface of the cement is touched. This shall be repeated at frequent intervals. The time of setting shall be the number of minutes elapsed from the starting of the mix to the time when the needle fails to make a perceptible circle on the surface of the specimen. The setting time shall be reported to the nearest minute.

H. ULTIMATE COMPRESSIVE STRENGTH.

H-1. A cylindrical mold 12 mm. (0.472 inch) high and 6 mm. (0.236 inch) in diameter shall be placed on a flat plate and slightly overfilled with cement of standard consistency. A second flat plate shall be pressed on top of the mold to remove excess cement. The molds shall be made of hard rubber, glass or other substance that will not be corroded by the cement. (Painting the molds with a thin solution of a hard wax in benzine will facilitate removal of the specimens.) Three minutes after starting the mix, the molds shall be transferred to an atmosphere of 100 per cent relative humidity at 37° C. (99° F.). Thirty minutes later, the specimens shall be immersed in distilled water at room temperature.

The ends of the cylinder shall be surfaced plane at right angles to the axis. The ends of the specimens may be ground flat by the use of a small amount of carborundum powder (200 mesh) and water. The molds containing the specimens are drawn back and forth across a glass plate coated with the abrasive and water. They should be rotated about one quarter turn every few strokes. The test specimens shall be kept wet during the grinding and until after crushing. The machine used in crushing the test specimens shall be operated at a rate of speed which will move the crushing head 0.25 mm. (0.01 inch) per minute.

The value for compressive strength shall be reported as the average of three or more from a lot of five specimens and shall be rounded off to the nearest 10 kilograms per square centimeter. If the values for individual specimens fall more than 15 per cent below the average, they shall be discarded and the average of the remaining specimens shall be reported. In case more than two of the specimens are eliminated, the test shall be repeated.

I. FILM THICKNESS.

I-1. A portion of a mix of standard consistency shall be placed between two flat square or round plates of uniform thickness. The surfaces of the plates between which the cement is spread shall be approximately 2 square centimeters (0.31 square inch). Three minutes after the mix is started, a load of 15 kg. (33 pounds) shall be applied vertically on the top plate.

Ten minutes after the mix is started, the thickness of the two plates with the cement film between them shall be determined. The difference in the thickness of the plates with and without the cement film shall be considered as the film thickness. An average of three tests shall be reported to the nearest 5 microns (0.0002 inch).

J. SOLUBILITY AND DISINTEGRATION.

J-1. The solubility and disintegration of a cement is a measure of the erosion plus the extraction of soluble material from the cement by the action of water.

One-half cubic centimeter (0.5 cc.) of cement of standard consistency shall be pressed between two flat plates until the cement disk is 20 mm. (0.79 inch) in diameter. A piece of fine platinum wire placed in the soft cement as the specimen is formed provides a convenient means of holding it. Three minutes after the mix is started, the plates and cement shall be placed in an oven at 37° C. (99° F.) in a relative humidity of

100 per cent for one hour. Two such specimens shall be used for each determination.

After one hour, the specimens shall be withdrawn from the oven, placed in a tarred weighing bottle and weighed. The combined weight of the two specimens and the weighing bottle, less the weight of the weighing bottle and the platinum wires, shall be taken as the weight of the specimens of cement. The specimens shall immediately be submerged by pouring 50 cc. of distilled water into the weighing bottle, which shall be stored for seven days at 37° C. (99° F.). The specimens shall then be removed from the water. There shall be no evidence of crystal growth or extensions from the surface of the specimen. The water shall be evaporated from the weighing bottle at a temperature just below 100° C. (212° F.). The weighing bottle shall then be dried at 149° C. (300° F.) to constant weight. The weighing bottle and contents shall be weighed. The difference between the final weight of the weighing bottle and its initial weight shall be the amount of disintegration. The gain in weight divided by the weight of the specimens times 100 shall be the percentage of disintegration. The average of duplicate tests (two weighing bottles containing two specimens each) shall be reported to the nearest 0.1 per cent.

K. PACKAGING.

K-1. The cement powder and liquid shall be supplied in properly stoppered glass containers. The net weight of the powder shall be indicated on the container. The liquid shall be supplied in an amount 20 per cent in excess of that necessary to combine with the total amount of powder when mixed to testing consistency.

K-2. Adequate and accurate instructions for proportioning and manipulation shall accompany each package. These instructions shall include the required temperature of the slab, the powder-liquid ratio, the rate of the incorporating powder and the time of mixing.

K-3. Each package of powder and liquid shall be marked with a serial number or a combination of letters and numbers which shall refer to the manufacturer's records for that particular lot or batch of cement powder or liquid.

K-4. The date of manufacture (year and month) shall be indicated on the package as a separate item or as a part of the serial number.

SUMMARY OF DETAIL REQUIREMENTS OF A. D. A. SPECIFICATION NO. 8

Consistency of Mix	Time of Setting at 37°C. (99°F.)		Ultimate Compressive Strength	Film Thickness	Solubility and Disintegration	Arsenic Content
	Minimum	Maximum	Minimum 7 days	Maximum	Maximum	Maximum
	Minutes	Minutes	Kg./Cm.²	Microns	Percentage by Weight	Percentage by Weight
Disk 30 ± 1 mm. in diameter	4	10	840 (lbs./in.²) (12,000)	40	0.30	0.0002 (1 part in 500,000)

A comparison of the foregoing revised specification with the current American Dental Association Specification No. 8 for Dental Cementing Medium[2] shows the differences, which for convenience are here itemized:

(1) *Title.* The title of the specification was changed to read "American Dental Association Specification No. 8 for Zinc Phosphate Cement." Because many types of cementing media are employed in dental technology, zinc phospate cement seems more appropriate.

(2) *Compressive Strength.* The one-hour compressive strength requirement was deleted. Cements which complied with the compressive strength requirement at one week and with the requirement for solubility and disintegration also complied with the compres-

sive strength requirement at one hour. The test is unnecessary and was eliminated to lighten the burden of testing.

(3) *Film Thickness.* The maximum value for film thickness was reduced from 50 to 40 microns. Values as low as 25 microns were suggested. Data collected in the testing of cements for compliance with the specification show that the value of 25 microns is too low. It does not permit a sufficient tolerance for a test of this type or for the manufacture of the cement.

(4) *Solubility and Disintegration.* The maximum value for solubility and disintegration was lowered from 1.0 per cent to 0.3 per cent. Several cements of current manufacture have a value well within the proposed maximum limit of 0.3 per cent. According to the data recently collected, there are cements having disintegrations as high as 0.6 per cent which will comply with the former specification. Cements of this kind will be eliminated by this change.

(5) *Arsenic Content.* The current specification permits one part of arsenic in 100,000 parts of cement (U. S. P.). The zinc phosphate cements on the certified list contained less than one part of arsenic in 500,000 parts of cement. The potency of very small amounts of arsenic in producing pulp death is well known. Therefore, the reduction of the arsenic to the lowest minimum is desired, and the maximum amount now permitted is only 1 part in 500,000.

(6) Items L-1 and L-2 (current specification[2]) pertaining to certain policies of the Research Commission were deleted because of the present testing program[6] of the Commission.

The foregoing specification will become effective July 1, 1938.

Manufacturers whose products meet this standard may guarantee and certify at any time that their cements comply with the revised specification.

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BUREAU OF PUBLIC RELATIONS

RELATIONSHIP OF PEDIATRICS TO PEDODONTIA

By Russell R. Hippensteel, M.D., Indianapolis, Ind.

THE word pediatrics, literally translated, means child cure. Likewise, the word pedodontia means child tooth. Thus we must realize that the pediatrician and the pedodontist stand on common ground—and on that common ground, in its idealistic sense, should have for their slogan the conservation, through prevention, of that which is most precious to the nation and to us, as professional men and women: the health of our children.

It is assumed here that the words pediatrician and pedodontist refer to those in our profession limiting their work to infants and children. Our work is general in scope and, since the majority of the nation's children must depend for health guidance on the general practitioners, it follows that we are in that group. Therefore, in this paper, the words physician and dentist will be used interchangeably with pediatrician and pedodontist.

Contemplation of this subject suggests:

1. Its unlimited scope. Many phases of our related work might be discussed, but because of lack of time, only a few of the problems of common interests will be taken up.

2. The fact that the pediatrician, being interested in the child as a whole, must include in his resources the services of the pedodontist, just as he does the services of the orthopedist, the oculist, the otolaryngologist and the surgeon.

3. My own limited ability to discuss your problems since I know so little about the technical side of your work—or is this lack of knowledge a qualification? In any event, I am not qualified to speak for the profession of pediatrics in general, and wish to state that any theories here advanced will be those advanced in research literature in these fields and those impressions gained in actual practice.

4. The realization, through actual experience, of the necessity for close relationships and frank discussions between our two professions, which must move on common ground since we are faced with the same diagnostic, therapeutic and research problems.

The necessity for a close relationship between our professions is, perhaps, more theory than fact. Most physicians realize how lacking they are in dental knowledge and interest; which prevents them from recognizing some of the most obvious dental conditions. On the other hand, too many dentists are equally deficient in sound medical practice in their daily work.

The prevention and treatment of any disease in childhood necessitates the utilization of all available recognized medical and dental aids. But, in actual practice, many of us have been confused and disappointed in the lack of cooperation between the two professions. Perhaps this is true because the pediatrician has too long considered the dental profession as one that employs merely mechanical technics and that should have nothing to do with the physical condition of the

(Read before the Section on Children's Dentistry and Oral Hygiene at the Seventy-Ninth Annual Session of the American Dental Association, Atlantic City, N. J., July 14, 1937.)

child beyond those conditions related to the teeth and the immediately adjacent structures.

Perhaps, in the past, some pediatricians have been justified in this attitude, but recently dentistry has made rapid strides, not only in technical skill but also in educational knowledge through broadened school curricula and requirements. If we pediatricians do not take cognizance of this increase in knowledge and do not recognize the interrelationship of our problems and those of the pedodontist, we must apologize for a shortsightedness that has blinded us to the usefulness of your profession.

These facts lead to a very pertinent question: How can a closer relationship between pediatrician and pedodontist be established?

A few points may here be enumerated:

1. Inclusion in the medical school curriculum of more useful instruction in the dental aspects of medical practice among children.

2. Frequent joint meetings between the medical and dental professions where problems of common interest might be discussed. These meetings would tend also to present a unified front to those who, outside our profession, talk of economic change and of our ultimate socialization.

3. Establishment of more preventive dental services in connection with children's hospitals, both private and public.

4. Correlation of those facts on which we are agreed and joint publication of medical and dental literature pertaining to children whenever such publications are intended for the education of the public.

A typical illustration of this need is a dental bulletin called "How to Build Sound Teeth." It sets forth a complete health and diet régime for mother and child. The only reference or concession it gives the pediatrician is by inference in the statement "When artificial feeding must be used, a special advisor should be consulted."

Such an attitude on the part of dentists naturally leads us into an uncooperative attitude and produces the question of whose duty, by virtue of training, it is to prescribe and regulate the dietary régime in children.

Obviously, it is the duty of the pediatrician. The pedodontist is rarely in a position to do this since his contact with the child comes much later.

Nor does the pediatrician have any objection to this procedure, provided the pedodontist is trained dietetically, understands the nutritional needs of a growing child, recognizes abnormal conditions other than those arising in the mouth and, most important, recognizes the methods of applying the diet to the particular type of child involved. It is not enough to have the dentist give fancy diet lists to hopeful mothers, without being able to discuss the various elements of an adequate diet, and the specific problems applicable to taking such a diet, especially those involving the psychologic aspect. We must remember that there is a vast difference in the reactions of children, and that upon such simple factors as explaining to a parent the difference between appetite and hunger rests the success or failure of any dietary routine. The problems then belong wholly in the realm of the pediatrician.

As to what constitutes an adequate diet for the child, no one short of a nutritional expert can determine. The great mass of evidence for or against the various dietetic theories appearing in our literature must be impartially weighed. This requires fundamental training and experience in the application of diets.

A basic diet, to produce normal growth in the child, should consist of thirty-eight separate and distinct food substances. Roughly, these are divided as follows: eighteen amino-acids, one fatty acid, twelve minerals, six vitamins, and some carbohydrates. How different this is from our ideas of a few years back! Then we were interested primarily in carbohy-

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drates, fats and proteins and a sufficient caloric intake.

Practically speaking, a child will, if he consumes a sufficient variety of foods, get all of these food elements. However, the over-emphasis on any one type constitutes a food fad and usually results in anorexia in the child. For example, there are the spinach advocates. Its value can be judged by this statement. It requires from 600 to 1,000 servings of this food to produce the amount of vitamin D in one teaspoon of cod-liver oil.

The recent work of Tisdall[1] has shown that, because from 30 to 50 per cent of a child's present caloric intake comes from purified carbohydrates, such as highly milled flours and sugars, our present-day standard diets are deficient in minerals and vitamins, especially vitamins B and D.

Every pediatrician has experienced the development of rickets and dental caries in one of two children fed exactly the same diet. This fact is only further proof that our knowledge of diets for proper growth is still in its infancy.

This discussion of the various elements necessary in the diets of children naturally leads to the question of their rôle in the problem of dental caries, which is the most serious local problem the pediatrician sees in children. The immediate problem is prevention, which comes within the scope of pediatrics. Its mechanical treatment and aids in stopping the process belong to the pedodontist. Theoretically, its prevention begins prenatally. Diets adequate in vitamins and calcium given the expectant mother should insure sound bone and tooth structure in the child. However, there is no evidence that this is true. The fetus, being a parasite, will take those elements from the mother without due consideration as to the supply in her body.

Apparently, there are two main schools of thought concerning the etiology and the prevention of dental caries. These two schools have produced a volume of literature almost equal to that of any other medico-dental subject. It is rather difficult in this literature to determine what is the product of pure research and what is not substantiated by fact.

The one school maintains that local conditions are responsible for the development of caries and that environment and heredity affect it; the other, that deficient diet and defective nutrition are responsible.

From the pediatric point of view, heredity may be a factor. Certainly no one will disagree with the statement that as far as caries being a purely local condition is concerned, the theory that "a clean tooth never decays" has failed to solve the problem, although it, no doubt, is a factor.

As to the second school of thought, based on the theory that dental caries is due to a dietary deficiency, most physicians will agree that an adequate diet is the greatest factor in its prevention and cure.

The pediatrician is keenly aware of the importance of the various dietary elements in obtaining maximum growth and development in children, especially in relation to the various vitamins and their functions. However, the effect of the application of these vitamins to laboratory animals does not allow one to assume like effects in the child. For example, vitamin C is necessary in infants to prevent scurvy, but is apparently unnecessary for its prevention in the animal.

Vitamin A is present in cod liver oil, leafy and yellow root vegetables and many yellow fruits. Its presence protects the animal against infection, but there is abundant proof, clinically, that it does not do likewise in the child. Its relationship to the teeth is questionable, although Howe has shown that it has to do with the function of the odontoblasts. Rarely is there clinical evidence of its lack in the well-balanced diet of the child.

Vitamin B occurs in wheat cereals and yeast. Its absence robs the rat of his appetite, but it is relatively insignificant

as a cause of anorexia in children, which every pediatrician knows is almost wholly on a psychologic basis. Likewise, it is reported to serve as a defense mechanism in its effect on teeth. Just what this means is not clear. Vitamin C is present in citric and certain other fruits and leafy vegetables. Hanke's[2] work on the importance of this vitamin on teeth tends to show that it is inhibitory in the production of dental caries.

Vitamin D is the antirachitic vitamin. It occurs in fish oils and egg yolk, also in sunshine and irradiated egosterol. It has to do with the metabolism of calcium and phosphorus, so important in the production of good teeth. An adequate supply of calcium and phosphorus from these sources will be utilized advantageously by the child if they are assimilated in the presence of a sufficient supply of vitamin D.

Important as these vitamins are, we must admit that our knowledge of them is still in a confused and elemental state, especially in view of the work of Boyd, Drain and Nelson[3] and Bunting,[4] whose work has shown that dental caries can be controlled and arrested by placing the child on a rigid diet of natural, well-balanced foods, plus good cod-liver oil, without the use of high vitamin concentrates and minerals. Therefore, a note of warning should be sounded regarding acceptance of the claims laid down by the sponsors of high concentrates of vitamins. The child's tolerance for vitamins cannot be overstepped in natural foods, but one cannot be too sure that synthetic and pure high concentrates have no potentialities for harm.

As Brenneman[5] has pointed out, it is well to be conservative about the use of such products and to await two desiderata in medicine: to adhere to what we know is good and not to go too far afield after strange gods.

Heretofore, the pedodonist has, perhaps, been more occupied with repair of the damage in caries than with its prevention. If the pedodontist is to help the pediatrician with the prevention of caries, it behooves him to acquaint himself with the fundamentals of the dietary requirements of children and their methods of application.

Both pediatrician's and pedodontist's beliefs about caries prevention and treatment will, no doubt, be influenced by their knowledge of these various theories set forth. However, another warning must be sounded: We must be careful to evaluate the theory and to remind ourselves constantly that there is a vast difference between the products of pure research and those faddist theories set forth in throw-away medical and dental journals which clutter our professional desks.

The tucking of these vitamins into bread, milk, cough drops, chewing gum and even face creams is advertising propaganda which strains our credulity. If physicians and dentists become credulous about these products, it follows that the public has sufficient reason to distrust our professions once the truth of our gullibility is exposed.

Infections in and about the mouths of children are common problems to both physician and dentist. One frequently confronting the pediatrician is the occurrence of alveolar abscess with its not infrequent involvement of the submental lymph glands. As to its treatment, we can have no disagreement. Early drainage of the infected area is necessary and important, if extensive cellulitis and, more rarely, osteomyelitis, are to be prevented. Intra-oral drainage of the abscess is certainly less destructive to the tissues. The question of removal of the offending tooth, especially if it is a deciduous one, is viewed differently far too often. The pedodontist seems to favor its retention to maintain tooth space. If the infection can really be eradicated by treatment, there is no objection. However, if it cannot, there should be no compromise and the tooth must be sacrificed.

Pyorrhea needs no comment since it rarely occurs in children.

Canker sores are frequently encountered. The cause is not known and the process is self-limited. These lesions clear up quickly with or without treatment.

One of the most confusing problems presenting itself to the pediatrician is that of so-called gingivitis, frequently called trench mouth, or various forms of stomatitis. Nearly all inflammatory lesions of the mucous membrane are generally thought to be due to the fusospirochetal organisms. There is much evidence that these organisms are only saprophytic; that is, they are the result of disease and not the cause of it, and that they thrive only when that tissue is devitalized.

The work of Lichtenberg,[6] which has thrown a great deal of light on this confusing problem, is much too extensive to be reported here.

In actual clinical practice, it is difficult to recognize in children so-called "trench mouth" infections. True it is that in many cases of acute gingivitis, characterized by inflamed bleeding gums, acutely sick children who have fever and with or without treatment recover in from five to seven days. In nearly all of these, Vincent's organism is present; but surely, since the condition is so acute and so self-limited, it cannot arise from these organisms.

Frequently, such symptoms are seen in children who have, because of one positive Vincent's smear, had a dental diagnosis of Vincent's infection. This is not justified except that, because this type of ulcerative stomatitis usually occurs in neglected mouths full of carious teeth, the resulting parental shock may serve as a stimulus to more frequent dental visits.

We must learn to speak the same language as to what constitutes Vincent's infection in the child.

The question whether infected teeth and their closely related structures serve as foci of infection in children is debatable. Of course, no pediatrician doubts that a child with dirty, carious, infected teeth will suffer nutritionally, but, speaking from a strictly clinical point of view, it would be hard to convince him that the poor nutrition is the result of a focal infection. It is more probably due to an inability to masticate properly.

Just as the medical field is dotted with therapeutic enthusiasts, so are there dentists who believe that rheumatism, heart trouble, eye infection and malnutrition can be improved or cured by dental means.

Clinical experience does not justify this enthusiasm, but whether the condition is local or focal, it goes without saying that full dental care should be given all children.

Another problem frequently encountered by both physician and dentist is that of malocclusion.

When pediatricians unite with pedodontists in attempting to detect and immediately treat incipient malocclusion, there will be less occasion for long tedious treatment later in life. We realize that this responsibility is ours since we have the greatest opportunity for observation early in the life of the child.

To this end, the pediatrician attempts to recognize the positive symptoms of malocclusion. From his standpoint, some of these attempts are listed as follows.

1. To correct faulty nutrition and prevent and properly treat the infectious diseases of childhood.

2. To recognize by the fifth year the absence of interproximal spacing.

3. To clear the nasal passages of all hypertrophied tissue to encourage nasal breathing.

4. To prevent, as far as possible, deforming habits, such as lip, thumb and finger sucking, and the use of pacifiers. (I am a little skeptical that these habits are the cause of as much deformity in children as dentists would lead us to believe.)

5. To refer to the dentists each child, by the age of $2\frac{1}{2}$ years, not so much for

actual treatment as to accustom him and his parents to regular dental visits.

If the physician could do this for every child, the responsibility for the detection of malocclusion would be the dentist's. This statement is the more evidently true since there seems to be a difference of opinion among dentists as to the age this treatment should be started.

One of the most important and common problems of physicians and dentists is the managing of children during treatment. A vast amount of literature based on psychology has been written on this subject. It is axiomatic that the possession of this ability spells success for both pediatrician and pedodontist. Much can be learned about it from written articles, but more often it is a native ability.

Children may be classified as motor and non-motor types. It takes little ability to handle the non-motor type. The motor type is sometimes difficult in his behavior and requires much patience.

A few rules might be enumerated:

1. To gain the confidence of the child, one must gain his interest by explanation.
2. Developed interest will be followed by cooperation.
3. Cooperation fails with falsehoods about pain, prolonged procedures and the use of force.
4. A firm, gentle, humane attitude, a genuine fondness for all children and a desire to treat the child, as well as his disease, will result in few failures in management.

Those in our professions not possessing these attitudes should not have children as patients.

CONCLUSION

It is hoped that some problems of common interest have been presented, together with evidence as to why there should be sympathetic cooperation between pediatricians and pedodontists.

From a common interest, we can learn a great deal from one another, to the mutual benefit of both us and our patients.

DISCUSSION

J. M. Wisan, Elizabeth, N. J.: The high point of Dr. Hippensteel's presentation is the statement that "dental caries is the most serious local problem the pediatrician sees in children." Thus, the relationship of the pediatrician and the pedodontist becomes a paramount issue for present-day health. Undoubtedly, an improved relationship of the professions will result in a decided improvement in dental health. If dental caries is the most serious local problem in child health (I question the use of the word "local"), dentistry merits a more prominent rôle than has been given to it in the past. We are, therefore, justified in hoping that the opinion of Dr. Hippensteel concerning the importance of pedodontia will be translated into reality. It would seem that until the dental profession is afforded more responsibility in public health programs by officials, physicians and educational administrators, and until private medical practitioners give to dentists the position which they merit in private medical practice, dental disease may continue to be a serious problem. Dr. Hippensteel is correct in suggesting improvement in the dental courses in the medical curriculum and in urging joint meetings of physicians and dentists. He asks for more attention to preventive dentistry in medical and dental literature. He also recommends that dental services in children's hospitals be instituted and hopes for a clarification of the causes of dental caries. All of these, he believes, will bring about an improved relationship between pedodontists and pediatricians. He speaks of increasing dental services in children's hospitals. Unfortunately, most hospitals stress emergency treatment only. My experience indicates that their results would be more beneficial if emphasis were placed on complete preventive treatment, including optimal diets, correction of enamel defects, orthodontic treatment for tooth and arch irregularities, routine roentgenography and mouth hygiene. Present-day nutritional knowledge does not, in most cases, assure dental health from dietary improvements unaided by the other preventive measures. The question of nutrition is one that leads to the suggestion: "To adhere to what we know is good and not to go too far afield after strange gods." If this principle is followed in dental care of

children, not only will we depend on optimal nutrition, but we will also provide careful preventive dental services and insist on thorough mouth hygiene. While it has been possible to place children on "rigid diets of natural well-balanced foods" within the walls of orphanages, I know of no one who has been able to state with any degree of certainty that in private practice it has been possible to attain strict adherence to an optimal diet for dental purposes. There seem to be too many environmental influences interfering with the provision of natural well balanced foods and too many temptations encouraging the harmful use of refined foods. Moreover, the practice of depending on dietary measures to arrest dental decay is a risky one. To my knowledge, there has been no proof that decay can be arrested by nutritional improvement alone for the average person in private medical or dental practice. The Iowa experiments, so often quoted, were made on too few children to warrant conclusive opinions. Furthermore, three questions asked of those conducting the Iowa studies, have thus far, to my knowledge, remained unanswered: 1. Were there any failures in attempts to arrest decay by nutritional improvement alone? 2. What was the percentage of success in arresting decay by nutritional improvement alone? 3. How is arrested decay diagnosed? Until these questions are answered unqualifiedly and scientifically in favor of arresting decay by nutritional measures alone, we must not place too much dependence on diet in treating dental caries. Dr. Hippensteel challenges dentists who refuse to extract untreated or improperly treated diseased deciduous teeth. I have discussed this question with many well-known dentists. Not one favored the retention of untreated or improperly treated diseased teeth. I have carefully read the dental literature and have failed to find one citation that recommends the retention of untreated or improperly treated diseased teeth. So there should be no disagreement on this point, particularly since mechanical space maintainers may be inserted in most instances. One point in Dr. Hippensteel's paper seems inconsistent with my studies; namely, that clinical experience did not justify the enthusiasm of dentists in believing that certain diseases can be improved or cured by dental methods. For example, Drs. Chenoweth and Selkirk state that tonsilitis, heart and kidney conditions and other diseases may arise from abscessed teeth, but also warn us that "the child has less resistance to infectious disease than has the adult," and "his blood serum contains fewer antibodies (except in the early months of life) with which to combat infection."[7] On this point, it will be well to consider the opinion of Russell L. Haden, M.D., who states[8]: "Failures in obtaining a cure or improvement in the condition due to focal infection by removing the focus should only stress more the importance of the early removal of dental infection. We should prevent the development of systemic disease rather than attempt to cure it after it has developed." From Dr. Hippensteel's previous remarks, I am confident he will substantiate these reports.

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DENTISTRY AT THE BOY SCOUT JAMBOREE

IF you were on a ten-day vacation, having the time of your young life, and you suddenly developed one of those galloping toothaches that you so often hear about, what would you do? Well, that's just what more than 150 Boy Scouts and their leaders in attendance at the first National Boy Scout Jamboree did. This monstrous convocation of over 27,000 of the youth of America assembled in the Nation's Capital at Washington, D. C., to live and play together for ten days developed toothaches, stomach aches and the little ills and pains that come to all growing boys; but, thanks to the forethought of Capt. Fred C. Mills, head of the health and safety program of the National Boy Scout organization, as well as others on the committee for the comfort, safety and health of these boys, the Scouts did not have far to go to get the relief they needed.

Naturally, in a city of more than 27,000 people, somebody is going to get a toothache, some piece of orthodontic appliance is going to get out of order, some porcelain jacket crown is going to be jarred loose by some of those sticky caramels that kids so dearly love—yes, even some extraction that should have been cared for before leaving for camp is going to demand action, and then the old heroic methods must immediately be put into force. No, it wasn't any fun to have to go to a dentist on this, their big vacation, but not in one single instance did any of these boys object to the treatment suggested by the dentists in charge.

Three dentists were assigned to the task of caring for this mobilized army that literally blew into the Nation's Capital and camped there in the shadow of the great Washington Monument for ten days.

These dentists were from different parts of the United States and were chosen for their interest in Scouting and their ability, along lines of dentistry, in handling boys: Dr. Raeburn R. Davenport, of Boston, Mass., a Scout commissioner and an instructor in the Harvard Dental School; Dr. Richard S. Cranmer of Camden, N. J., a Scout Master in his home town, and Dr. Irving A. Clark, of Janesville, Wis., a specialist in children's dentistry and the holder of the Silver Beaver, the highest award given to adult Scouters for meritorious service to boyhood. Working together in perfect unison, these three members of the dental corps were on twenty-four hour call and ready to meet any emergency or care for any dental operations that might arise. Fortunately, no major casualty did arise.

A regular army equipment was provided and the dental tent was one of the finest in the entire tented city. A wood floor and side wall covered by a regulation army tent made an ideal dental office, even if the old army chair was a bit stiff and the old type of foot engine was as much a relic as some of the things the boys went to see in the Smithsonian Institution. Many of these boys, from twelve to fifteen years old, had never seen a foot engine, and even some of the older leaders marveled at the dexterity that was shown by these operators in "throwing her into high" and pumping away at an offending molar. Thanks to the efficient service of the United States Army and the supply department of Walter Reed Hospital under the capable direction of Tom Hoover, an Army man, the dental unit was well supplied and kept equipped with all the necessary articles to run a first class office in any city. Everything that was asked for was cheerfully supplied; which went a long way toward making this unit a success.

International dentistry, by the way, was also indulged in by the three dentists in charge. Two cases from Poland, two from Canada, one from Bermuda and two from

England were charted on the report. In conversation with these and others from foreign nations, it was learned that the governments of other nations more or less make it their business to see that dental care is one of the main objectives in the health and safety of their people. That, of course, is not true of our own country, and although these Scouts and their leaders were supposed to have had a thorough check up on their health condition before they came to the Jamboree, it was clearly shown that the examination and follow up work required for the Jamboree was inadequate, and it was the recommendation that hereafter when a national encampment of the youth of this country is to be held, a thorough dental examination and follow up treatment be required by a registered dentist before a boy is allowed to register for such an encampment. The dental unit apparently sold themselves to the Health and Safety Committee of the Boy Scout organization, but nevertheless such conditions as were found in from 80 to 85 per cent of these boys should have been checked by the home town dentist before they left for their camp.

It was a thrilling experience to the three dentists who were appointed to the dental unit and many new and lasting friendships were made. Several dentists of national reputation stopped in to visit and add a word of encouragement to those in charge. It was a thrill to have Dr. C. Willard Camalier, the President Elect of the American Dental Association, drop in on his way to Atlantic City, where he was to take over the Presidency in a few short days. Dr. E. H. Bruening, of Omaha, the only dentist on the Health and Safety Committee of the Boy Scout organization, got busy with his candid camera and spent an hour with his sleeves rolled up "shooting" a few of the patients and the doctors.

Dr. Leslie W. Foster, the field dentist for the Indian Reservations under the United States government care, and F. Regadas, a dental student from Mexico City, were interested visitors. A. V. Gibson, of Belfast University, Ireland, also visited the camp.

DENTAL HEALTH FOR AMERICAN YOUTH

Washington, D. C., Dental Society's Resolution

October 26, the Washington, D. C., Dental Society passed the following resolution endorsing the American Dental Association's program of Dental Health for American Youth. Copies of the resolution were sent to key public health and educational officials. It is suggested that every state and component dental society follow the example set by the District of Columbia dentists.

RESOLUTION OF THE WASHINGTON, D. C., DENTAL SOCIETY

Whereas, mouth health is a vital factor in the incidence of general health, and is so recognized by the medical and dental professions;

Whereas, the American Dental Association adopted for its meeting held in Atlantic City in 1937 the slogan "Preventive Dentistry in the Interests of Health"; and this year, the theme "Dental Health for American Youth," for the purpose of more thoroughly educating parents and children in preventive measures designed to minimize the dental diseases of youth in the present and future generations; and

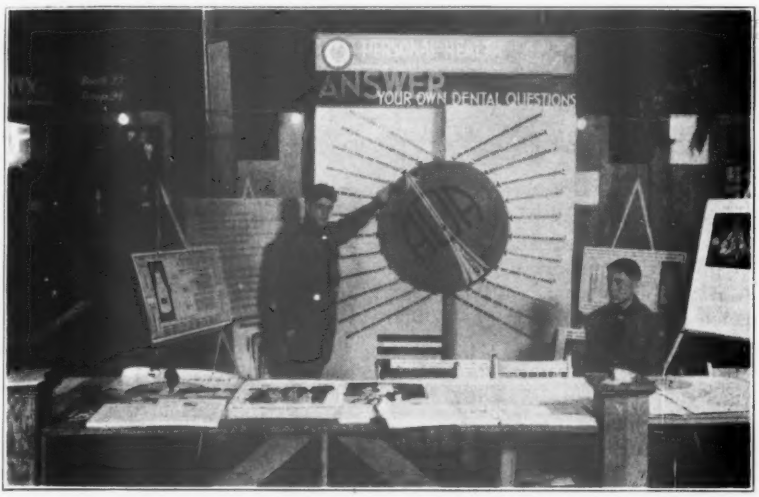
Whereas, the District of Columbia Dental Society, a constituent of the American Dental Association, recognizes the extreme importance of this move in the interests of the children of Washington and vicinity; be it

Resolved, that the District of Columbia Dental Society enthusiastically endorses this program of the American Dental Association

and pledges its wholehearted support in the carrying out of the purposes thereof; and be it further

Resolved, that a copy of this resolution be forwarded to the commissioners of the District of Columbia, the health officer, the president of the board of education, the superintendent of schools, the District of Columbia Medical Society, the Surgeon General of the United States Public Health Service and the American Dental Association.

Daniel F. Lynch, *Secretary-Treasurer,*
District of Columbia Dental Society,
Washington, D. C.



San Francisco District Dental Society booth on personal health, Boy Scout campaign, Municipal Auditorium, San Francisco, April 1937.

Exhibit of the Thomas W. Evans Museum and Dental Institute, School of Dentistry, University of Pennsylvania, at the Atlantic City meeting. This exhibit won first award in Division B.

DENTAL ECONOMICS

EDUCATING OURSELVES TO EDUCATE THE PUBLIC

By George Wood Clapp, D.D.S., New York City

DENTAL education of the public is extremely important. It is important to people generally because it will help them to avoid and overcome disease and may lengthen life. It is important to all dentists collectively because only by such education can dentistry win public esteem as an outstanding form of health service.

It is important to you and me because with it we can be more successful than we can without it.

In order to develop as promptly and forcibly as possible the intensely personal and practical reasons that each dentist should be an intelligent and continuous educator in his community, let us confine this study to the relation between the dental education of the public and the success of the individual dentist. We can then study some of the methods of public education most likely to make the individual practitioner successful.

WHAT CONSTITUTES SUCCESS FOR A DENTIST?

Because no two of us are alike, it is natural that our definitions of success should differ in details. There are three things necessary to success on which we may be able to agree, at least for this study: We all want comfort in the present, sweetened by some luxury. We all want security in sickness and old age. We all want happiness to grow out of our work as a flower grows out of the ground, since without happiness there is no success.

The dentist cannot attain two of these objectives—comfort and security—unless he is financially successful. They are economic objectives and must be paid for with money. Happiness is an emotional objective and is to be won only by a liking for the work and a perception of its importance in the lives of other people. That dentist is successful, in the terms of this study, who, by competent and conscientious service, earns for himself and his dependents comfort, security and happiness.

IMPORTANCE OF FINANCIAL SUCCESS

Financial success is only one of the foundation-stones of all-round success. It has very important qualifications and distinct limitations. At least a moderate amount of it is necessary to professional success. It will not make a dentist either competent or honest, but it makes it easy to be both. It cannot, alone, bring happiness to any except the selfish heart.

There are probably many in this audience to whom any definition of success that does not begin with the patient's need and end with the dentist's obligation will come as a shock. They were brought up, as I was, to believe that the dentist's success was measured solely by the clearness with which he perceived the patient's need, and the skill with which he served it. It was assumed that if he saw clearly and served well, he would

(Read before the Section on Periodontia at the Seventy-Ninth Annual Session of the American Dental Association, Atlantic City, N. J., July 14, 1937.)

automatically be successful without special attention to the financial side of practice. Things do not work out that way. Service and success are not necessarily companions, and the only way the dentist can keep them together is to step between them and walk hand in hand with both.

Let me bring you another profound conviction from years of study. If you will follow your investigation fearlessly to the end, it will make no difference whether you begin your study of the dentist's success with the dentist or with the patient. Starting with either, thoroughness and honesty will bring you to the conclusion that the service of the financially unsuccessful dentist is a poor investment from the patient's point of view. The intelligent patient wants and tries to procure good service and is willing to pay a fair fee for it. The financially unsuccessful dentist is usually incapable of rendering good service. Worry gives him a wrong mental attitude. Poverty keeps him from postgraduate courses and good equipment. His need may force him to be dishonest with his patients.

ARBITRARY STANDARDS OF SUCCESS

During the writing of this paper, there lay in plain sight a rule marked with two units of measurement, inches and millimeters. Both units are arbitrary devices to meet urgent needs, and both, when well understood, are very useful. Our units for measuring success in dentistry are arbitrary units to meet special needs. One is a money unit; the other, a time unit.

The money unit is the sum of money the dentist should earn annually over office expenses in order to be competent and honest in his work and comfortable and secure in his own life. The time unit is in two parts. The first part is the number of years during which he earns the money unit. The second part is a closing date by which he should have provided for his own old age and for his family if he dies.

For the money unit, I use the sum of $5,000 annual earnings, net over office expenses, as the threshold of success. That is a minimum in cities. It may be more than is necessary in small communities.

For the first part of the time unit, the dentist should be earning $5,000 a year by the close of his first five years in practice, and, given good health, he should earn at least that much for each of thirty years. As to the closing date of the time limit, he should have at least a modest competency by the age of 55, which is thirty years after graduation; but this is by no means final. Many a dentist takes on a new lease of life after 55 and does well.

HOW MANY DENTISTS ARE SUCCESSFUL?

How many dentists are successful according to the money and time units?

Let us take the history of some general practitioners from the report of the Committee on the Study of Dental Practice.

During the first five years after graduation, these dentists averaged a net income of $3,000. During the second five years, they averaged $4,700, and this is very nearly the most that two dentists in every three will ever earn. During the third five-year period, one-third of these dentists earned enough to raise the average of the whole group to $5,800. This average is more or less fictitious, because only one general practitioner in three in the group ever reached it. At the end of the third five-year period, when the dentists were about 40 years old, a decline in income began and was continuous and progressive, but it was difficult to trace because of individual differences and many deaths.

What do these figures mean? That it took these dentists thirteen years to get to our threshold of success. That is nearly three times as long as I think it should have taken if they had been properly trained for practice. They mean that two dentists in every three never got across that threshold; which is, to me, a very severe arraignment of our system of education. These dentists held their top level

of net income only about five years, which is only one-sixth as long as they should have been able to hold it if they had good health. Just when they had really learned something about dentistry and were of maximum value to the public, they started down the other side of the financial hill and had less and less chance to accumulate a competency.

MEASURING SUCCESS BY PURCHASES

Let us look at another group of dentists concerning whose gross and net incomes no figures are obtainable. There are in Manhattan, the Bronx and Westchester County, New York, about 4,000 general practitioners. Five in every ten of these 4,000 get along with about one dollar's worth of supplies a day, exclusive of precious metals, and are so unsuccessful that it does not pay dental depots to mail them advertising matter or send salesmen to call on them. In three of every five of these practices, the dentist is without credit even for the dollar and supplies can be sold to him only on a C.O.D. basis. Of the remaining five in every ten, about half are visibly successful and the other half hover between failure and success.

We may close this part of the paper by saying that two dentists in every three apparently never cross the threshold of success as I have defined it, and that they reach their maximum earnings much later than they should and hold them a shorter time than they should.

WHY ARE NOT MORE DENTISTS SUCCESSFUL?

In a vocation which offers service of unexcelled importance to the public, which is protected from outside competition and which has all the materials for a more interesting, important and persuasive story than any commercial business can offer, why are so few dentists successful? Two conditions have done more than all other things combined to prevent success: a mistaken mental attitude and insufficient education of the undergraduate for practice. Let us examine the former first.

A MISTAKEN MENTAL ATTITUDE

About seventy years ago, some of the better type of dentists rebelled against the methods of other dentists. They rebelled, not against their knowledge and skill, but against the trickery and quackery in their business methods. They grouped themselves together under the general title "ethical dentists," or, as we might say, "square-deal dentists"; for ethics is merely the square deal in action. It was an emotional reaction, and, naturally enough, the pendulum, which had been away over on the side of quackery, swung too far to the side of self-righteousness.

These ethical dentists renounced and denounced all connection between any business methods and the conduct of a dental practice. Their first great error was to rule out the motive of self-interest on the part of the dentist as unethical. They believed that the dentist's only obligation was to the patient of the moment, never to himself or to his own family. They considered it unethical to know what service cost or to quote in advance the fee for a given operation, even when asked or urged by the patient. It was unethical to send out bills more than once a year, and then only as a gentle hint that some money would be useful. Patients were allowed to pay if, as and when they pleased, and many never pleased.

This mental attitude has caused thousands of tragedies in the lives of dentists, who, but for it, might have gone far. The only dentists who have achieved success are those who have broken away from it and developed for themselves a psychology of success fully as ethical in theory and far more ethical in practice.

INSUFFICIENT EDUCATION FOR PRACTICE

The other hindrance to success is insufficient education for practice. The dental student is under the impression that he is being educated for practice. He

is not. He is being educated for one-seventh of practice, theory and technic. There is, generally speaking, nothing approaching adequate provision in the dental educational system for training the student for practice as a whole; and as far as that system goes, he will remain unconscious of those entirely different things on which six-sevenths of his success will *have* to rest, until he finds himself in practice and out of step with the world. Unfortunately, the awakening may not come until late in life.

Something is afoot to correct this disastrous deficiency in dental education. In 1935, the American Association of Dental Schools published "A Course of Study in Dentistry," which proposes to start the student aright in his relations to the other healing professions, to make him understand the place and function of dentistry in the community and to apply sound principles of ethics and economics to his practice and his personal life.

This material, excellent as it will be for the dental students of the future, does not and, I suppose, cannot get down to an honest recognition of two facts which underlie this whole matter of education and practice: the reasons that men choose dentistry and why they practice it.

WHY DO MEN CHOOSE DENTISTRY?

If I were to believe what I sometimes read and hear, I should think that men choose dentistry in response to a divine call; that into the souls of a few selected high-school graduates there comes such a vision of the grandeur of dental service that, forsaking all else, forgetful of self, they give themselves to dentistry as missionaries go to foreign fields.

It simply is not so. That is not because the men who have chosen dentistry had lower ideals than other men. They were moved by the common motive of self-interest and by the desire to engage in a profession that should lead to comfort, security, public esteem and happiness.

I have asked thousands why they chose dentistry. Very few chose it for dentistry itself. Some were brought up in a professional atmosphere; some loved the fine mechanics; some thought it a road to social distinction; some believed it an easy way to earn a lot of money. In every case, we come back to the motive of self-interest. There is no harm in this, if we remember that a selfish expression of self-interest cannot bring happiness.

Dentistry is capable of justifying the better aspirations of these men. There could easily be plenty of practice for every competent and conscientious practitioner. Dentistry gives, to those of us who are moderately successful, lives that are luxurious in comparison with many of the lives around us. No other form of health service is more important, and none would be more acceptable to the public, if it were rightly understood. The trouble is not with dentistry itself: it is with a mental attitude and education that make dentistry difficult to practice and unattractive to the public.

NO ADEQUTE EDUCATION FOR PRACTITIONERS

Even if we could have fearless honesty in undergraduate instruction, beginning now, that would not solve the problems of those of us who are in practice and cannot return to college. There is no authoritative source in the dental educational system to which we can go for instruction in these important fundamentals. We shall have to go to outside sources for this education. Fortunately, each of us can educate himself if he cares to take the trouble.

WHY SHOULD THIS PAPER CONSIDER THESE THINGS?

Perhaps you are saying to yourself, "Why bring these things up?" Because, before we shall be capable of educating the public, we must educate ourselves, and we cannot do this unless we are prepared to be fearless and honest. Who do you think is to blame for the fact that half the

dental offices in the United States are half empty of patients? Do you think it is the public? I can assure you that it is not. The only thing that keeps dentistry from enjoying high esteem is that we are too lazy to wake up to our opportunities and improve them. When you want to find out why an office is half empty, ask the dentist to look in the mirror. The reflection will show the man who is to blame in that he is out of step with his opportunities and his obligations. When that man gets back into step with these fundamentals, there will be no trouble about patients.

AMONG THE SUCCESSFUL DENTISTS

Let us now spend some time with the two or three dentists in every ten, the country over, who have crossed the threshold of success, and some of whom are far beyond it.

Observation shows that these successful dentists have developed, each for himself, a surprisingly uniform mental attitude, very different from that I have described, and that each has imposed on himself a course of training in those nonprofessional elements of practice upon which 85 per cent of success must rest. The closer the observation, the stronger grows the conviction that their success is due to this attitude and self-training. Let us see what they have done:

They have recognized that self-interest is the dominant power in every life. Self-interest is only the modern name for the old biologic principle of self-preservation. It is tremendously important because it furnishes motive and will-power in the struggle for success. These dentists have discovered that it is suicidal for the professional man to seek his own welfare at the expense of the welfare of his patients, and that every incompetent or dishonest word or act is a nail in the coffin of success.

They have discovered that dentistry is not an end in itself for any one. For them, it is the means that they have chosen to earn comfort, security and happiness. For their patients, it is a means to good looks, comfort, employability, economy. The ends these patients seek—that practically the whole public seeks—rest on good health. These dentists are students, servants and teachers of dentistry in relation to good health. They are not merely white-collar repair men.

They have found that dentistry as a health service has a story which can be made interesting, important and persuasive, and that when it is told as people want it told, they will listen. And so they have mastered one of the biggest tasks of their lives: They have learned to tell that story as people like to hear it.

So much for their mental attitude. Now look at their office administration: They know their costs, and they expect a fair profit over those costs except in charity and study cases. Their fees are suited to the financial levels of those whom they serve. In some offices, the fees are moderate; in some, they are high; but they always insure to the patients competent and conscientious service.

In short, they have mastered the four great principles on which all successful business rests: a good service, an honest, interesting and persuasive story about that service, unswerving integrity and good value. The wiser among them take a keen enough interest in children to keep the practice full.

What is the net result of this attitude and these methods? These men score a higher percentage of success for a longer time than any other dentists. They are winning for dentistry such public esteem as it has. During the depression, the public evidently divided dentists into two groups, the servants and teachers of health in one group, the repair men in the other. People abandoned the repair men in great numbers, so that their practices fell away from 60 to 100 per cent. They stayed with the servants of health as long as they could, and, generally speaking, these dentists suffered far less from the depression than any others.

WIDER EDUCATION OF THE PUBLIC

Valuable as is chairside education of visitors to dental offices, it is nothing like sufficient. Not more than one dentist in three is capable of it. Probably not more than one adult in ten of those who can afford to be private patients gets it. If the other 90 per cent of desirable people will not come to us, we must go to them and interest, convince and motivate them where they are and as they go.

There are four important ways of reaching the public: (1) newspapers and magazines; (2) public addresses; (3) the radio; (4) the public schools. Let us look at them, remembering that we must reach the same people, not merely once or twice, but many times for repeated and progressive instruction.

Newspapers and magazines care very little about dental articles except news articles. These appear only occasionally, so that they do not build an audience. They do not usually carry any lesson, and they have very slight educational value. Public addresses, when given by skilful speakers, undoubtedly have value, but the average dental talk, before a luncheon club for instance, is uninteresting and deadly. This leaves us only the radio and the schools, and it is to these that the balance of this paper is devoted.

THE RADIO AS A MEANS OF REACHING THE PUBLIC

Many dentists believe that if they can get a dental program on the air, good results are bound to follow. No idea could be more mistaken. Success on the radio is hedged about with invisible but unyielding requirements, and these are trebled for a dental program, which is inherently one of the least interesting of all programs and one of the most difficult to make successful. Here, more than anywhere else, the public can and does tune the uninteresting speaker out promptly and conclusively.

The text matter must be interesting to the public from the public's point of view. The dentist's conception of its importance avails practically nothing, so that the more eminent the dentist, the deadlier the program is likely to be. Merely dental knowledge avails little here.

The broadcasts must be frequent over a long period. The dental program that, with two or three broadcasts a week, builds a regular audience in two years is doing very well indeed. Incidental broadcasts have only accidental and very slight value.

THE APPROACH THROUGH THE PUBLIC SCHOOLS

The schools are the best doorway by which to get a radio program into homes. Much good work is being done in the schools. The American Dental Association is doing excellent work within the limits of its personnel and budget. Other agencies are doing well also.

Within the area between New Haven, Conn., and Harrisburg. Pa., it has been possible to make a nearly ideal combination of radio talks in printed form as lessons in the schoolrooms and as broadcasts over the air for the benefit of parents.

The requests for printed broadcasts as lessons have spread far outside the radio area, and these lessons are used each week in more than 6,000 schoolrooms in twenty-seven states from the Atlantic to the Pacific and from the Lakes to the Gulf. When they are put on as mimic broadcasts in the rooms, these talks stimulate pupils to undertake a program of spontaneous physical self-correction as nothing else has ever done. C. Carroll Smith, famous for his work in the schools of Peoria, considers them the most helpful method of teaching dental health in his experience. Bridgeport, Conn., reports that 4,000 more pupils turned in good dental-health cards this year than in any previous year and gives the radio program credit for much of this showing. That means a lot of visits to local dentists. These children send their elders to dentists. One class of thirty pupils sent

thirty-one of the adult members of their families to dentists for ninety-one operations.

We can apparently have as many schoolrooms as we are willing to earn. Concerted action by dentists generally would bring a weekly audience of a million children. In Indiana, as the result of activities initiated by Dr. Westfall, dental health educator, 100,000 children in 3,275 schoolrooms, in 401 communities, received one lesson a week for thirteen weeks from the printed broadcasts last spring. It is hoped to give similar lessons for twenty-six weeks during the next school year. That will be equivalent to more than 2,600,000 individual lessons in one state in one year.

Unlimited opportunities are before us. They call for our best talents and unlimited application. If we neglect them, they will pass us by. We shall be paid for our neglect by public indifference toward dentistry and by conditions in the profession much as they are now, when two dentists in three are never successful enough to justify the time and money they spent for a dental education.

SUMMARY

There is no more highly privileged group of workers in America than we are. There is great public need of our teaching and service. Millions of people await only education to become regular patients at fees fair to themselves and to us. We are protected from outside competition, and that protection is being steadily increased. A dental diploma and license should go far to guarantee success to any competent and conscientious dentist within the modest limit that we have drawn.

An acquaintance has just written an interesting chapter in his life. He graduated from dental college forty-three years ago at the age of 22. It took eight years for him to discover the errors and shortcomings in his mental attitude and education. He immediately undertook a rigorous course of self-training, which has been continuous since that day. He has been a competent and conscientious dentist, in no way famous, unknown except for the people who trust him and whom he serves. His fees have always been moderate.

He has lived comfortably, but thriftily, with about the same ups and downs as the rest of us, except that his savings account has never been neglected and he has invested instead of speculating. He has found time to develop hobbies in which he is keenly interested. At the age of 65, he has retired from practice, bought a modestly luxurious home at Miami Beach and has about $100,000 worth of other available assets which produce a living income.

Something closely approaching this should be possible to thousands of dentists, granted good health.

Compare the achievements of dentists generally under circumstances at least as favorable. Two dentists in three never get across the threshold of a modest success, and two in three of those who get across stay there only a short time. The public is uninstructed as to the tremendous contribution good dental service can make to almost every life. Thousands of people whom we might do much to save are failing physically every year or dying prematurely.

The dental educational system contains no provision for training us for all of practice. And when some one points out our opportunities and suggests that we go to work to improve our condition, we are apathetic. We are among the greatest living examples of "Let someone else do it."

New facilities for self-training are now available. Any dentist who will invest a few dollars a year and a few hours of time in the education of his community can begin to develop dentally conscious audiences that will do much to change this condition of public ignorance to one of public understanding and appreciation.

He can also begin to impose on himself the training necessary to get him across the threshold to success.

The substance of this paper is that education of the public must begin with education of ourselves and that it calls for alert and continued interest and effort on our part. In return, it offers the inducement of success to every competent and conscientious member of the profession.

REPORT OF THE MEETING OF THE AMERICAN PUBLIC HEALTH ASSOCIATION

More than 3,800 members attended the 1937 meeting of the American Public Health Association held October 5 to 8 at the Pennsylvania Hotel in New York City.

In previous years, dentistry has been fitfully represented at the American Public Health Association meetings. This year, preventive dentistry permeated practically all of the sessions.

Four papers on different phases of this problem were delivered: "Indiana's State-Wide Program of Oral Health," by Howard B. Mettel, M.D., and Mary H. Westfall, D.D.S.; "Evaluating Dental Health Education and Dental Service programs," by J. M. Wisan, D.D.S.; "Lactobacillus Acidophilus and Dental Caries," Philip Jay, D.D.S., and "Some Facts About Nutrition and Dental Caries," by Bion R. East, D.D.S. In addition, the importance of preventive dentistry for children was stressed by several other essayists in other sessions.

C. Willard Camalier, President of the American Dental Association, addressed the members of the American Association of School Physicians at their annual banquet Sunday evening, October 31. On Monday, the American Association of School Physicians voted to change their constitution so that all school health workers may become members. The new name of the organization is now the American Association of School Health. Plans are already under way to invite all school dentists and public health dentists to join.

Thursday, October 7, a luncheon meeting of the oral health group was held at the Pennsylvania Hotel. Those present included forty-five dental members of the American Public Health Association and others from fifteen states and Canada. John Oppie McCall was appointed chairman of the group for the coming year and Harry Strusser was appointed secretary. They were instructed to prepare a program for next year's meeting at Kansas City.

During recent months, there has been some discussion regarding the advisability of recommending the establishment of a dental section in the American Public Health Association. It was the consensus of opinion of those present at the oral health luncheon that the time was not propitious and that both dentistry and the other health groups would be better served if the dental members affiliated themselves with the existing sections. All agreed that more dentists should belong and take an active part in American Public Health Association affairs.

In the exhibit section, dentistry was represented by exhibits from the Bureau of Public Relations of the American Dental Association, the Guggenheim Foundation and the United States Public Health Service. A motion picture prepared by the Guggenheim Foundation and entitled "Modern Dental Care" was shown throughout the meeting. Several trips to New York health centers and to the New York City Health Departments enabled visiting dentists to view the work being done for children by the Dental Division of the New York Department of Health.

COUNCIL ON DENTAL THERAPEUTICS

ACCEPTED DENTAL REMEDIES

The following articles have been accepted as conforming to the rules of the Council on Dental Therapeutics of the American Dental Association. The Council wishes dentists to understand that the admission of an article does not imply a recommendation. A copy of the rules that govern the Council in the consideration of articles will be sent on request.

SAMUEL M. GORDON, *Secretary.*

PHENETIDINE, AMINOPYRINE AND SALICYL COMPOUNDS[1]

Aspirin Tablets, 5 grains-Abbott: A brand of Tablets of Acetylsalicylic Acid, N.F.

Manufactured by the Abbott Laboratories, North Chicago, Ill. No U. S. patent or trademark.

Aminopyrine Tablets, 5 grains-Abbott: A brand of Tablets of Aminopyrine, N.F.

Manufactured by Abbott Laboratories, North Chicago, Ill. No U. S. patent or trademark.

Aminopyrine Tablets (Aromatic) 1 grain-Abbott.

BARBITAL AND BARBITAL COMPOUNDS[2]

Phenobarbital Soluble-Abbott, 1½ grain Tablets: A brand of Tablets of Soluble Phenobarbital, N.F.

Manufactured by Abbott Laboratories, North Chicago, Ill. No U. S. patent or trademark.

COD LIVER OIL AND RELATED SUBSTANCES[3]

Abbott's Cod Liver Oil: It has a potency of not less than 1,500 units of vitamin A per gram and 100 units of vitamin D per gram.

Manufactured by Abbott Laboratories, North Chicago, Ill. No U. S. patent or trademark.

PHENOLIC COMPOUNDS[4]

Solution Cresol Saponated-Abbott: A brand of Saponated Solution of Cresol, U.S.P.

Manufactured by Abbott Laboratories, North Chicago, Ill. No U. S. patent or trademark.

SOAP[5]

Liniment of Soft Soap, U.S.P.—Linimentum Saponis Mollis (Lin. Sapon. Moll.) An alcoholic solution containing about 65 per cent of soft soap, perfumed with oil of lavender.

1. A.D.R. 1937, p. 170.
2. A.D.R. 1937, p. 69.
3. A.D.R. 1937, p. 94.
4. A.D.R. 1937, p. 176.
5. A.D.R. 1937, p. 193.

Liniment Soft Soap-Abbott: A brand of Liniment of Soft Soap, U.S.P.

Manufactured by Abbott Laboratories, North Chicago, Ill. No U. S. patent or trademark.

DENTIFRICES[6]

Curtis Tooth Paste: Composition: See Sheffield Tooth Paste (A.D.R. 1937, page 110).

Manufactured by the Sheffield Company, New London, Conn. Distributed by East Lake Drug Company, Minneapolis, Minn.

6. A.D.R. 1937, p. 105.

CURRENT LITERATURE

A Comparative Study of the Innervation of the Periodontal Membrane

By W. Lewinsky and D. Stewart

The innervation of the periodontal membrane of human and animal teeth was investigated. Serial sections stained by Cajal's method were employed. It was found that the end-organs, which always lie toward the peripheral part of the membrane, were associated with the thicker type of fiber. In carnivora a single thick convoluted fiber was found. In rodents a coarse spider-like nerve ending appeared. Insectivora showed only fibers terminating in knob-like swellings and in this resembled the structures found in man. What is probably a second type of ending was found in all types of mammals. This was in the form of fine fibers which showed looping and dichotomy.—*Proc. Roy. Soc. Med.*, 30: 1355, 1937.

B. G. Bibby.

Dental Hypoplasia

By Ch. Ruppe and Henault

The authors studied the diagnosis of dental hypoplasia on a chronological basis on 487 children at the Hérold hospital. Ninety children (20 per cent) had dental hypoplasia. An investigation of 50 children seems to show a higher proportion among heredosyphilitics. Contrary to the usual statement, hypoplasia of the deciduous teeth was frequently found in connection with congenital syphilis. The character of the hypoplasia in the deciduous teeth was the same as that in the permanent but less systematic. In the permanent teeth, on the other hand, only 12 cases of isolated hypoplasia were found out of 56, the distribution being more generalized in the rest. Only 4 cases of Hutchinsonian teeth were found out of 67, two unilateral and two bilateral. A diagnosis of the cause of the hypoplasia may be made in most cases, but with caution; for sometimes teeth are unaffected when chronologically they should have been, as shown by the neighboring teeth.

In 65 cases where an etiological investigation was made, heredosyphilis was found as the cause in 33, nothing in 20, and in 13 there were various causes such as eclampsia, whooping cough, typhoid, rickets, myxedema, and bronchopneumonia.—*La Revue de Stomatologie*, December 1936; per Abstract *Den. Rec.*, 57:566, October 1937.

Virgil D. Cheyne.

Chemical Constitution of Enamel and Dentin

By W. D. Armstrong and P. J. Brekhus

Analyses on enamel and dentin separately from a number of teeth gave the following data: enamel, calcium -35.41 ± 0.96, phosphorus -17.45 ± 0.51, magnesium -0.30 ± 0.05, carbon dioxide -3.00 ± 0.25; dentin, calcium -26.18 ± 0.34, phosphorus -12.74 ± 0.48, magnesium -0.83 ± 0.08, carbon dioxide -3.57 ± 0.10, nitrogen -3.36 ± 0.15.

Since the magnesium and carbon dioxide contents of dentin are higher than those of enamel, it is concluded that the mineral phases of enamel and dentin are not identical.

Enamel from carious teeth is not different from that of sound teeth. There is no correla-

tion between composition of enamel and susceptibility to decay or with age of eruption. As wide variation is found in the composition of enamel in different teeth from the same mouth as in teeth from various mouths.—*J. Biol. Chem.*, 120:677, 1937.

H. C. Hodge.

Rate of Apposition of Enamel and Dentin Measured by the Effect of Acute Fluorosis

By Isaac Schour and Henry G. Poncher

The effects of injection of small doses of sodium fluoride on growing teeth were studied in a hydrocephalic infant between the ages of 5 and 9 months. The dose varied from 1 to 2 cc. of a 2.5 per cent solution. The medical history and observations made at autopsy showed no general toxic effect of fluorosis. A histologic analysis yielded the following observations:

The dentin showed a stripe of disturbed calcification and the enamel showed a stripe of disturbed growth in the portion of the tissue which had been forming and calcifying at the time of each injection.

In the deciduous teeth, a distinct ring of disturbed calcification was found in the enamel and the dentin which had been forming and calcifying at the time of and soon after birth. These rings are regarded as neonatal rings and constitute a landmark which separates the prenatal from the postnatal portion of the deciduous tooth. The histological analysis indicated better prenatal than postnatal calcification.

Tables are given showing the rate of apposition of enamel and dentin, with doses of the sodium fluoride and the intervals between injections.—*Am. J. Dis. of Child.*, 54:757, 1937.

Frank Allen.

Relation of Dental Focal Infection to Ocular Diseases

By V. L. Jones

The author states that "it is often difficult to persuade the dental surgeon to be as radical and at the same time as conservative as we consider justifiable in view of the many ocular conditions that have been relieved by the removal of dental focal infections." The literature on dental focal infection is reviewed and the complexity of the problem emphasized. A case of the author's is presented to show the relationship between the teeth and ocular infection. In focal infection involving the eye, the first consideration is the teeth; second, the tonsils; third, the paranasal sinuses; and then the prostate, gall bladder, appendix, etc. The danger of wholesale roentgenographing by improperly trained individuals is deplored and called to the attention of the dentist and physician.—*J. Missouri M. A.*, 34:293, August 1937.

Hamilton Robinson.

Diagnostic Importance of the Tongue in Internal Medicine

By B. I. Comroe

"Were the tongue carefully inspected in every patient reporting to the dentist or physician, important diagnostic pointers would frequently be found." Color, fur, pallor, papillae, evidence of inflammation, blood-vessels on the under surface, form, size, movement and humidity of the tongue should be considered. Many individuals, especially smokers, may have a coated tongue even in good health. The state of the tongue in various abnormalities and diseases is given.—*Am. J. Med. Sc.*, 194:661, November 1937.

Hamilton Robinson.

Glossodynia

A physician from Louisiana wrote to the editor of Queries and Minor Notes Department of the *Journal of the American Medical Association*, inquiring for a diagnosis and treatment for a patient with the following history:

"To the Editor:—I have a patient who complains of a sensitive tongue. He is a white man, aged 40, and is in good health otherwise. His tongue appears normal to inspection and his teeth and gums are in good condition. There are no sharp edges to irritate the tongue. The condition has been present for four years. He has remissions and exacerbations of the disease. He states that the tongue feels scalded as if from drinking something too hot. Hot foods and highly seasoned foods make the pain worse. Smoking seems to make the condition worse and he has omitted tobacco for months at a time but still has the trouble. The pain is located on the tip and sides of the tongue. His nose and throat are normal. Gastric analysis gives normal results. He does not have indigestion. There is no cardiorespiratory disease. There are no al-

lergic symptoms. The Wassermann reaction is negative. The red and white blood cell count is normal."

Answer.—The diagnosis should be glossodynia, for the description mentions no sign of inflammation of the tongue. Glossodynia may be due to (1) ill-fitting dentures pressing on the anterior and posterior palatine foramina, (2) disturbance of the temporomaxillary joint by an abnormal bite, (3) sensitization to artificial dentures, (4) unclean fissures in a scrotal or lymphangiomatous tongue, (5) electric currents between metal dentures, (6) disease of the lingual tonsil, (7) avitaminosis, (8) pernicious anemia or (9) idiopathic glossodynia.

Only causes 5 to 9 need be considered in this case.

5. Lain in 1932 called attention to the presence in many mouths of small electric currents, which in some instances can be stopped by replacing tooth fillings so that all will be of the same metal. (Lain, E. S.: Chemical and Electrolytic Lesions of the Mouth Caused by Artificial Dentures, *Arch. Dermat. & Syph.* **25**:21 [Jan.] 1932; Electrogalvanic Lesions of the Oral Cavity by Metallic Dentures, *The Journal,* March 11, 1933, p. 717.)

6. Sluder in 1918 described the effects of the disease of the lingual tonsil. Pain at or near the tip of the tongue is one of those effects. (Sluder, Greenfield: Some Clinical Observations on the Lingual Tonsil Concerning Goiter, Glossodynia and Focal Infection, *Am. J. M. Sc.* **156**:248 [Aug.] 1918.)

7. Avitaminosis, possibly related to pernicious anemia, is sometimes responsible for pain in the tongue. (Hutter, A. M.; Middleton, W. S., and Steenbock, Harry: Vitamin B Deficiency and the Atrophic Tongue, *The Journal,* October 21, 1933, p. 1305.)

8. Pernicious anemia often causes pain in the tongue even before the blood or stomach signs have appeared or there is any inflammation of the tongue. The case under discussion, if no other cause of the trouble has been found, should be watched with this possibility in mind.

The pressure of tumors or an inflammatory neuritis may cause glossodynia in rare cases. Cancerphobia has something to do with some cases, though it is often a result of the pain rather than a cause of it. Discovery of a cause amenable to treatment makes a good prognosis.

9. There are cases, however, in which the cause remains hidden. For these palliation is the only possible line of treatment and this is usually unsatisfactory, for anesthesia destroys the sense of taste. It may be attempted by having the patient hold Dobell's solution in the mouth for several minutes, or by the use of ethylaminobenzoate."—Queries and Minor Notes, *J.A.M.A.,* **109**:1297, October 16, 1937.

M. K. Hine.

Radiographic Differential Diagnosis

By H. M. Worth

Interpretation demands recognition of normal and abnormal appearance, and determination of cause. Clinical and roentgenographic findings must be coordinated.

In senile atrophy the alveolar socket is absorbed more regularly. In any case of pyorrhea the condition becomes more serious if rarefaction has preceded the marginal resorption.

Normal teeth have unbroken periodontal membrane and *lamina dura.* Anatomical land marks and bone spaces must be considered in apical osteitis. Watch the lamina dura in all cases. When sclerosing osteitis is found at the apex of a pulpless tooth, it is probably due to disease, but may appear normally anywhere. In productive periodontitis deposition is on the tooth side of the periodontal membrane. An unerupted tooth shows enamel, dentin and pulp chamber. Composite odontomas are inclosed in fibrous capsules. Salivary calculus can be recognized by an occlusal view showing it to be lingual. Anatomical projection of bone will also appear as such on an occlusal view. The above points are not true of sclerosing osteitis.

Apical foramina are funnel-shaped if growth is arrested before completion of the root. The foramen is convergent in rarefying osteitis.

A cyst appears as an area of bone destruction with no structure in it, having a clearly defined and cortical margin. Dentigerous cysts have organized tooth substance in them. Multilocular cysts may not have a well-defined bony cortex and have gross trabeculae. An osteoclastoma does not have the well-defined cortex and has delicate trabeculae.

Fractures are diagnosed roentgenographically by breaks in continuity of bone. The apparent line of solution must not continue

past the bone margin.—*Brit. D. J.*, **63**:389, 1937.

L. H. GARRISON.

Dental Disease and Affections of the Eye

By S. WHITNALL

CORRELATION between dental disease and affections of the eye has gained in practical significance only since William Hunter, in 1910.

Infections of the mouth may infect the eye, *Streptococcus viridans* being the most common. No part of the eye is immune, the most frequently involved parts being the iris, ciliary body and choroid. Paths of infection are either along the bones (direct), or through the blood or lymph channels (indirect). It has been suggested that the perivascular lymphatics may transmit infections to the general circulation, and then to the eye. Indirect infections can originate in either jaw and affect both sides, chiefly the eyeball. Direct infections arise in the upper jaw alone. They affect the orbital structures outside the eyeball on the same side.—*Brit. D. J.*, **63**:412, 1937.

L. H. GARRISON.

Stomatitis Due to Vitamin B_2 Deficiency

By W. AYKROYD and B. KRISHMAN

THE authors observed stomatitis cases, which suggested fungus infection, in groups of school children in South India. The typical case described showed white patches at the angles of the mouth extending to the mucous membrane of the tongue, with the sides and tip being generally involved, the anterior portion of the buccal cavity being red and eroded while the posterior portion along with the larynx was free of involvement. In the investigation as to the cause, the diet was thought to be at fault, as was proved when vitamins A and B were excluded and when vitamin B_2 was not. The deficiency disease described has still questionable factors as to whether the condition is of a multiple deficiency etiology or due to various constituents of the vitamin B_2 complex.—*Ind. J. Med. Res.*, **24**:411, October 1936.

B. J. FREY.

Anoxia in General Anesthesia

By M. D. NOSWORTHY

THIS is one of a series of articles which deals with the underlying causes of the emergencies that arise during the administration of general anesthetics.

Anoxia, or lack of oxygen, is the underlying factor in many anesthetic troubles. It manifests itself by irregular pulse rate, depressed breathing, nausea, muscular twitching and cyanosis. Pyrexia, fear, pain and hyperthyroidism all increase the patient's oxygen requirements and must be given consideration in our technic of administration.

Carbon dioxide has come to be considered the respiratory stimulant of choice. Individual susceptibility to carbon dioxide varies greatly, and if respiratory stimulation following its administration is not immediate, its use should be discontinued at once or toxicity results.

Overdosage of preoperative sedatives tends to make the patient a hazardous anesthetic risk, as they are prone to depress the blood pressure and respiration to a dangerous level and make the administration of anesthesia hazardous.—*Brit. M. J.*, **63**:809, October 23, 1937.

J. VOLKER.

Dietary Requirements in Pregnancy and Lactation

By G. C. M. M'GONIGLE

THE reproducing female requires not only sufficient dietary constituents for her own bodily maintenance, but also additional foodstuffs in order to nourish her offspring *in utero* and at the breast. Restricted diets below the basic requirements should never be recommended. There is evidence to support the view that it is food restrictions or deficiencies that are potent causal factors in toxemias. Also, the idea that special diets are essential for the reproductive phase of female activity does not coincide with physiological dicta. It is necessary merely that a variety be included to cover all essentials and a sufficient quantity to satisfy caloric requirements. Gross shortage of calories, except among a very small section of the impoverished, is rarely encountered. In such shortages, the deficiency is frequently corrected by carbohydrate feeding, and this is unfortunate because the basal requirement balance is thrown off. There is, as yet, lack of reliable data as to the optimal intake of fats, but it appears that certain ones are essential for bodily health. Protein frequently is found to be low. On advised diets protein is recommended to be at least 2 gm. per kilogram. This is to insure coverage of essentials. Deficiencies of minerals are quite common.

Anemia seems to be a common complication of iron deficiency. There is no evidence that excess of iron is harmful. Calcium is another mineral which is likely to be deficient. Milk is one of the main sources of it. Sufficiency of vitamins has been very hard to determine. Here again the margin of safety is advised. Favorable results have been reported in administrations of A and D.

During lactation it is well to maintain the essential diet with plenty of protein.

Pharmaceutical preparations are discouraged, as the food eaten should contain sufficient amounts if balanced. There is some reason to believe that the natural products may be better than those prepared by artificial means.—*Brit. M. J.*, **63**:259, August 7, 1937.

VIRGIL D. CHEYNE.

An Analysis of Cardiac Irregularities Produced by Calcium, and Their Prevention by Sodium Amytal

By H. E. HOFF and L. H. NAHUM

INTRAVENOUS injections of calcium salts are considered a routine clinical treatment in various pathological conditions. It has also been noted that cardiac irregularities sometimes follow their administration. Experimental work with laboratory animals confirmed the clinical observation that intravenous calcium injections produce cardiac change. It was further noted that sodium amytal in narcotic doses suppresses these symptoms and prevents cardiac arrest.—*J. Pharm. & Exper. Therap.*, **60**:425, 1937.

J. VOLKER.

Lympho-Sarcoma Cell Leukemia

By RAPHAEL ISAAC

IT has been observed frequently that blood of patients with lymphosarcoma may become leukemic. This syndrome has been characterized as leukosarcoma by Steinberg or lymphosarcoma terminating in lymphatic leukemia. The lymphosarcoma cell in the blood stream is usually mistaken for a lymphocyte. This type of cell may constitute 4 to 98 per cent of the leukocytes in the peripheral circulation. Some of the visible signs at the onset of the disease are: visible tumors in the neck region, sore throat, submaxillary enlargement, joint pains, fever and bleeding. On necropsy it is found that all the lymphoid tissue is transformed in the lymphosarcoma type.—*Ann. Int. Med.*, **11**:657, October 1937.

E. HAMPP.

Abscess of Lung

By L. S. T. BURRELL, M.D.

IN the lung, abscesses may be single or multiple and result from a variety of conditions. A considerable proportion of single lung abscesses are seen following tonsillectomy or some other operation on the nose, throat, and teeth. The first symptoms are a feeling of malaise and sometimes a bad taste in the mouth. The breath is often offensive even before there is any sputum, the temperature is raised and there is not free drainage through a bronchus. It may be hectic in type. If there is sputum, it may contain elastic fibers and pyogenic organisms and is not usually offensive unless there is gangrene, which is said to be due to the Miller-Vincent spirochetes present in the gums of many normal adults. Abscesses of the lungs should always be regarded as very serious lesions and should be handled as such.—*Brit. M. J.*, **63**:789, October 23, 1937.

E. HAMPP.

A Modification of Mallory's Connective-Tissue Stain, with a Discussion of the Principles Involved

By GERMAIN CROSSMAN

IN proposing the present modification of Mallory's connective-tissue stain, the author emphasizes the advantage of controlling the staining and destaining processes by means of a microscope rather than time standards or judgment; also the incorporation of the orange G with acid fuchsin rather than with anilin blue, which lessens the amount of dyestuff needed and more consistently stains erythrocytes and myelin yellow.—*Anat. Rec.*, **64**:33, August 1937.

E. HAMPP.

A Remarkable Lichen Planus Lesion of the Tongue

By D. W. MONTGOMERY

THE author was consulted by a woman, 76 years old, who had a papular eruption on the backs of her hands. She was in good general health. A tentative diagnosis of *lichen planus hypertrophicus* was made. At this time there were no oral lesions. Four months later the patient returned with a large, oblong, fluffy, white mass situated far back on the median

line of her tongue. Anterior to this mass were a few scattered, circumscribed, soft, dull white, flattened papules 2 mm. in diameter and a characteristic lacelike patch. There were also two small white papules in the pouch of the right cheek. A week later the large lesion had undergone involution and the smaller patches subsequently formed a leukoplakia-like coating over the dorsum of the tongue. The condition became progressively acute. Labial lesions developed, first as innumerable red dots, evidently congested capillary loops. These loops later became obscured by a white coating and then erosion set in with pain as the chief symptom. From the observations made at the beginning of lichen planus on the lips in this case the author believes capillary congestion to be the first change. The large, fluffy white mass which made an early appearance on the tongue was the result of hypertrophy of the filiform papillae incited by lichen planus. If the same condition were pigmented it would be diagnosed as black, hairy tongue.—*Arch. Derm. & Syph.*, 36:833, October 1937.

HAMILTON ROBINSON.

Frenum of Upper Lip

To the Editor: A child, aged 11 months, has a separation of the upper central incisors due to hypertrophy of the frenum of the upper lip, which is attached to the roof of the mouth. At what age should this muscle be cut and at what place? Should the teeth be wired now or should one wait until the permanent teeth appear?

M. D., Arkansas.

Answer. In general the best time for operation on the frenum labii superioris is between 5 and 6 years or between 9 and 10 years. Separation of the central incisors either temporary or permanent is not in itself an indication for operation. The medial suture between the intermaxillary bones normally persists in the anterior portion until quite late in life. The central incisors, therefore, both temporary and permanent, normally erupt with quite a wide space between. The permanent central incisors are normally brought in contact by the pressure of the erupting lateral incisors and canines.

There is a wide range of variation within normal limits in the structure and attachment of the frenum. Usually at birth or some time afterward it is attached at the crest of the alveolar process. As the alveolar process grows downward and forward, carrying with it the temporary incisors, the attachment of the frenum should be left behind. It occasionally happens that the frenum extends over the crest of the alveolar process and is attached to the fibrous tissue of the incisal papilla on the palatal slope. In such cases the attachment is not left behind on the labial surface of the process but extends between the incisor teeth as a pad or mat of fibrous tissue, which prevents the central incisors from coming together. This condition cannot usually be positively determined before the fourth or fifth year. When operation is indicated, this dense mat of fibrous tissue should be dissected out from between the teeth and removed, the wound being closed by two or more sutures in the gum tissue.

When operation is delayed until the ninth or tenth year, the teeth should be moved together by a mechanical appliance first and then the appliance removed; the teeth will separate in a few days. The operation is then to be performed, the tissue dissected out and the appliance replaced and the teeth immediately brought together so that in healing the connective-tissue fibers will unite from tooth to tooth as they should. This band, extending from the mesial surface of one central incisor to the other, normally holds these teeth in contact and unless this attachment is formed immediately after the operation the teeth will eventually separate. Operation before the completion of the temporary denture is seldom if ever indicated.—Queries and Minor Notes. *J.A.M.A.* 109:974, September 18, 1937.

Disorder of Taste Sensation

To the Editor: May I ask what line of inquiry you would suggest in attempting to make a diagnosis in this case? A woman, aged 55, single, complains of a disagreeable taste in the mouth, constantly present. She cannot enjoy any food or drink because the tongue perceives practically nothing except the bad taste. The tongue feels swollen along the left side, which margin looks slightly swollen and has enlarged veins. These veins would probably not have been noticed except for the patient's complaint. At first I thought it was a soreness she felt, and when the usual treatment with antiseptics and so on failed to improve it, I sent her to her dentist. He

also can find no condition to account for the symptoms. Outside of the tongue, I have found nothing to blame. There is no pressure on the veins at the base of the tongue. The condition began five or six weeks ago. The past week she stated that the other side began to feel the same way, but I cannot see what is doing it. This is a very incomplete question, I realize, but can you suggest what line of inquiry should be started?

M. D., Illinois.

Answer. There are multiple factors which might influence the nerves supplying the sense of taste. So little information is given in the inquiry as regards the lines of clinical or laboratory investigation which have already been applied that it is difficult to suggest any specific course. It is well known that pathologic changes in the area of the uncinate gyrus of the brain will affect the sensory nerves of the tongue. (Prinz, Hermann, and Greenbaum, S. S.: Diseases of the Mouth and Their Treatment. Philadelphia: Lea and Febiger, 1935, p. 510).

Prominences of the sublingual venous supply are common in mouth examination, though usually symptomless unless the condition becomes angiomatous with compression. Certain types of anemias, more especially pernicious anemia, are frequently first discovered by a glossy, sensitive tongue with areas of denuded epithelium, but rarely will the sense of taste be seriously affected.

More common causes of bad taste are imperfect cleansing of pockets or sepsis of the oral cavity. Certain drugs when taken at regular intervals, or even occasional doses, more especially nerve sedatives, basic salts or narcotics, may cause temporary unpleasant sense of taste. Obstruction by calculi of the salivary glands, inflammation, or radiation treatments over the sublingual ducts may cause considerable disruptions of salivary secretion and taste.

Since the patient is 55 years of age she is extremely fortunate if she does not have one or more artificial dentures or restorations. Chemical and metallic tastes that come from basic denture material such as sulfur, or coloring products found in latex-vulcanite dentures are often a source of bad taste as well as the rubber mouth odor. Most of the newer proprietary dental base materials contain such elements as phrenolresin, ammonia, camphor, or nitrocellulose, which almost without exception will give the wearer an occasional unpleasant taste.

During recent years much research has been done on the possibility and symptoms of electrogalvanism between dissimilar and variously alloyed metallic materials used in dental restorations. Case reports of such cases are now common in both medical and dental literature. A taste of copper, zinc, silver, aluminum, mercury, nickel or phosphorus, as well as irritation of the tongue even to the degree of erosion and ulcers, may occur as a result of electrolysis through the saliva between the metallic restorative dental materials.

A more elaborate and explicit aid in the diagnosis of the patient's trouble may be obtained by reviewing the following bibliography:

RATTNER, HERBERT: Stomatitis Due to Sensitization to Dental Plates. *The Journal,* June 27, 1936, p. 2230.

LAIN, E. S.: Chemical and Electrolytic Lesions of the Mouth Caused by Artificial Dentures. *Arch. Dermat. & Syph.* 25:21, January 1932.

LAIN, E. S.: Electrogalvanic Lesions of the Oral Cavity Produced by Metallic Dentures. *The Journal,* March 11, 1933, p. 717.

MACDONALD, W. J.: Chemical and Electrogalvanic Burns of the Tongue. *New England J. Med.* 211:585 (Sept.) 1934.

SCHWANKE, W.: Disturbance of Taste by Influenza. *Klin. Wchnschr.* 15:93, January 18, 1936.

LAIN, E. S., and CAUGHRON, G. S.: Electrogalvanic Phenomena of the Oral Cavity Caused by Dissimilar Metallic Restorations. *Am. Dent. J.,* September 1936.—*J.A.M.A.* 109:1063, September 25, 1937.

PRACTICE IN PARAGRAPHS

This department is intended for busy readers. It aims to tell a vivid story in a few words. Items should be of a practical character, and free from padding—not more than a paragraph or two. We exact no limit, but two hundred words can tell most of these stories. Send in your ideas; they will be welcome.

Cow-Horn Forceps—Pro and Con—With the cow-horn forceps one can remove morphologically normal mandibular first permanent molars with less tissue destruction and trauma than with any other instrument available. They should, however, never be used on deciduous teeth. The forcing of the horns into the bifurcation of deciduous roots means sure injury to the bicuspids. Thus, an instrument despised by many operators has its advantages when used properly in the right place.—*James A. Matthews, Glen Cove, N. Y.*

Explorer—Preventive dentistry is not being practised fully enough to be effective. In examining three thousand school children for fourteen consecutive years, I am convinced that the dentist is still only a decay chaser. He is modern enough to use bite-wing films to discover hidden caries but he fails to correct developmental defects which can only be discovered with a very fine explorer in the good old-fashioned examination technic. One can only practise preventive dentistry by actually preventing decay. Plenty of fine, sharp explorers should be kept handy and the supply replenished frequently, as they wear out rapidly if used properly. If this is done, many so-called perfect mouths will offer plenty of opportunity to practise ideal preventive dentistry.—*James A. Matthews, Glen Cove, New York.*

Matrix Inconsistency—The matrix is an invaluable adjunct to gold foil procedures. It has met with disfavor justly when its uses have been abused. If amalgam or a wax pattern can be adequately manipulated with a matrix, so can foil filling, when the technic is tempered with judgment. When it is placed for a distoclusal cavity in a bicuspid, for example, the foil should be so welded as to cause forced separation. This enables one to establish a contact point after removal of the matrix. Its use is seldom indicated in a mesioclusal cavity or in any other place where direct access is obtainable. The protection afforded the cervical gum tissues is another distinct advantage, giving a sense of security against slipping instruments. Experience teaches that its abuse rather than its use has placed it in disfavor. A stretch of one's imagination may place a distoclusal matrix cavity in the Class I division regarding accessibility.—*Herbert Ely Williams, Red Bank, N. J.*

Office Dentifrice and Polishing Agents—An easily prepared and comparatively inexpensive dentifrice for office use may be prepared from pumice and glycerin. Flour of pumice is mixed into a paste with glycerin (U.S.P.), and flavored with a few drops of some essential oil. Glycerin costs about 30 cents a pint, and flour of pumice about 40 cents a pound. A few cents' worth of some essential oil will flavor a year's supply. The matter of mixing is simple. Place about 4 ounces of flour of pumice in a clean serving bowl or soup dish. Add and stir glycerin into the mass until it becomes of the consistency desired. Then add a

few drops of some essential oil, such as peppermint, wintergreen or any other desired, to suit the taste. This will make about 5 ounces of dentifrice, which lasts about two months, and which costs only about 16 cents. It may be kept in a jar in the cabinet and used in amounts desired from a small dappen dish. An excellent polishing agent can be made similarly by using powdered chalk in place of the pumice, or stannic oxide may be used. It is quite easy to make suitable combinations by incorporating pumice in the polishing pastes according to the degree of coarseness or fineness desired. The most popular of similar dentifrices and polishing agents costs the profession about one dollar for a somewhat smaller quantity, and I have found that my patients like the home-made preparation much better.—*Albert D. Alexander, Medical Arts Bldg., Norfolk, Va.*

Trauma—Trauma has been more successfully publicized as an excuse for neglecting gold foil than as a contributor to the loss of teeth. When single-rooted teeth have carried large foil fillings for fifty or sixty years, the posterior teeth, with their two or three strong bracing branching roots, should not even be mentioned as potential victims of gold foil trauma. Much sustaining evidence proves that foil-filled teeth often outlive, by many years, intrinsically sound ones, which are lost from pyorrhea or other causes. If unbelievable migratory movements with orthodontia appliances cause no trauma, reasonably-sized malleted foil fillings should have a mild stimulant effect rather than acting as a detriment. Most loose teeth are lost from lack of vigorous mastication, as they are designed and planned for extensive overwork. The enlargement of the bony structure supporting the teeth of teeth-hanging trapeze performers substantiates a fact that offsets many well written, well meaning theoretic claims. Practical proof generally outranks theoretic contentions.—*Herbert Ely Williams, Red Bank, N. J.*

Sequelae of Decayed Deciduous Dentition.—Of course deciduous teeth are temporary, but they are the guides of permanent teeth—orthodontically, of course; but another equally important consideration is the control of caries. Decay of a first permanent molar on the mesial surface often is caused by direct contact with decay of the second deciduous molar. If decay is not present at the time of contact with the deciduous tooth, invariably the enamel surface is etched, and the carious process may be so slow that even a decade might pass before its destructive force is apparent. This is true not only of the lower first molar but of the upper as well. Then too, as the cuspid is usually the last anterior tooth to erupt, the decayed deciduous cuspid may be in contact with the lateral incisor or even the bicuspid. This can be simultaneous with either the maxillary or submaxillary dentition. It is not uncommon at all to have a bicuspid tooth of either the upper or the lower dentition erupt and make a contact with its adjacent deciduous tooth, which may be decayed, and thus incipient caries, or at least etching of the contacting enamel, may result. The principle involved is that a "bad apple touching a good apple invariably injures the good apple." You cannot safely store good apples in contact with decayed ones. Decay of the deciduous teeth can be the cause of decay of twenty permanent teeth. As astounding as this may seem, it is a fact. The lateral incisors, the cuspids, the bicuspids and the first molars of the permanent teeth are at the mercy of the decayed deciduous teeth. Yet there is a means of prevention, the proper filling and care of these temporary teeth.—*Esther E. and F. J. Svoboda, Cozad, Nebr.*

BIBLIOGRAPHICAL

"Juvenile Dentistry"

By WALTER C. MCBRIDE, D.D.S., F.A.C.D., director of the Department of Pedodontics, School of Dentistry, University of Detroit, Detroit, Michigan. Second Edition, thoroughly revised: Illustrated with 247 engravings. Philadelphia: Lea & Febiger. 1937. Price $6.00.

This second edition of McBride's book on juvenile dentistry is the response of the author to the enthusiastic reception of the first edition by the large group in the profession who are particularly interested in children's dentistry. Because of the prominent part which Dr. McBride has taken in the evolution of this phase of dentistry into practically a distinct specialty, the name McBride has almost become synonymous with the term children's dentistry.

The profession and particularly the children of the country owe much to the energetic enthusiasm of the author in reviving and promoting an aspect of dental practice that, several years back, was becoming more and more neglected by the profession generally. The problems and difficulties peculiar to juvenile dentistry have diverted many good and conscientious practitioners from practice on children, and this book is in substance a detailed account of the methods and practices employed so successfully by the author in an effort to promote interest in children's dentistry.

Dr. McBride devotes considerable attention to the features of children's dentistry that have made juvenile dentistry so unattractive, even impossible, to some, with many valuable suggestions for child management; and with invaluable suggestions and measures designed to eliminate the pulpless and abscessed teeth, deciduous as well as permanent. In fact, the book is the record of the experience in the application of methods evolved by the author over a period of fifteen years.

Elaboration of the contents of the first edition has evoked the addition of several new chapters by collaborative authors; namely, "Morphology of the Deciduous Molars, with Special Reference to Pulp Cavity and Its Relation to Cavity Preparation," by James Nuckolls; "Root Resection," by C. Taylor Hall, and "Physical Diagnosis" by H. V. Dwyer; all of which constitute an invaluable addition to this popular work.

"Principles and Practice of Public Health Dentistry"

By J. A. SALZMANN, D.D.S., editor of the *New York Journal of Dentistry;* head of the dental service of New York City Vocational Schools; member of the New York Society of Orthodontists, American Public Health Association, Etc. With a foreword by Alfred Walker, D.D.S., F.A.C.D., member of New York State Board of Dental Examiners; associate professor of New York University College of Dentistry; formerly chairman of the Committee on Community Dentistry, New York Tuberculosis and Health Association. With a special chapter by John Oppie McCall, A.B., D.D.S., F.A.C.D., F.A.A.P., director of the Murry and Leonie Guggenhein Dental Clinic; chairman of the Dental Advisory Committee of the Committee on Neighborhood Health Development, New York City, and by Harry Strusser, D.D.S., chief of the Dental Division, New York City Department of Health; lecturer on Dentistry, Columbia University College of Dental and Oral Surgery. Boston: The Stratford Company. Price $4.00.

Dr. Salzmann, in this book, has given us a virtual "*omnium gatherum*" of the principles and practice of public health dentistry. He has, with infinite detail, gathered together under one cover the complete developmental history of every phase of public health dentistry. In fact, as the history of this subject is so intimately related to general dental history, this book furnishes, in its way, a most interesting and fairly complete history of dentistry, but, of course, confined to Dr. Salzmann's particular phase of the general subject.

The enormity of the task of gathering so much material and recording the references

to that which is not included here is quite apparent, and the results of the effort elicit only praise from those accustomed to search of the literature.

The author has presented a well-amplified record of the development of oral hygiene, the position of the dental hygienist and dental disease prevention evolving from the development of oral hygiene. The story of oral hygiene is most interestingly told and in a manner satisfying in completeness but not overburdened with minutiae.

Dr. Salzmann has given much attention to dentistry as a recognized factor in public health and has kept in close touch with the social trends that have impelled the inclusion of dental practice in the scheme for social security, and here presents a critical analytic study of all the factors involved.

The book logically divides itself into four parts: Part I. The Evolution of Preventive Dentistry, wherein is included a sketchy but exceedingly interesting history of preventive dentistry as it is being evolved. Part II. Organization of Dentistry and Public Health, in which is included a history of organized dentistry and a discussion of the events which led to the development of dentistry as an autonomous profession; together with a history of the development of the work of the dental hygienist and the dental assistant. Part III. The Rôle of Official and Unofficial Public Health Agencies in Dentistry, in which are considered the origin of public health service and the relation of federal and public health agencies to dentistry. Part IV. The Dental Public Health Program, in which the author presents a review of all the activities public and private that have led to consideration of social health security from the standpoint of governmental administration.

Dr. Salzmann has also incorporated in the book valuable collaborative contributions by Dr. Harry Strusser, who is in charge of the community dental health program being carried out in New York City; and by Dr. John Oppie McCall, director of the Guggenheim Dental Clinic in New York City.

This is a most complete and elaborate study of every phase of public health dentistry that will prove of much value as a reference on this subject; one of the most helpful features of which is an extensive bibliography of the literature at the end of each chapter.

BOOKS RECEIVED

"A Textbook of General Biology"

By E. GRACE WHITE, Ph.D., professor of biology, Wilson College, Chambersburg, Pa. With 336 Illustrations. Second revised edition. St. Louis: The C. V. Mosby Company. 1937. Price $3.00.

"Berichte erstattet am IX. Internationalen Zahnarztekongres der F. D. I." (Transactions of the Ninth International Dental Congress, Vienna, 1936.)

Wien, 2. bis 8. August 1936. Erster Band 1. Halfte: Sektionen I bis VII. Verlag Urban & Schwarzenberg, Wien und Berlin.

"Essentials of College Chemistry"

By G. H. WHITEFORD, professor of chemistry; and R. G. Coffin, associate professor of chemistry, Colorado State College. St. Louis: The C. V. Mosby Company. 1937. $4.00.

"The Management of Fractures, Dislocations, and Sprains"

By JOHN ALBERT KEY, B.S., M.D., St. Louis, Mo., clinical professor of orthopedic surgery, Washington University School of Medicine; associate surgeon, Barnes, Children's, and Jewish hospitals, and H. EARLE CONWELL, M.D., F.A.C.S., Birmingham, Ala., consulting orthopedic surgeon to the Tennessee Coal, Iron & Railroad Company, and the Orthopedic and Traumatic Services of the Employees' Hospital; associate orthopedic surgeon to the American Cast Iron Pipe Company; attending orthopedic surgeon to the Crippled Children's Hospital, St. Vincent's Hospital, South Highlands Hospital, Hillman Hospital and Children's Hospital, Birmingham, Ala. Member of the Fracture Committee of the American College of Surgeons, American Academy of Orthopedic Surgeons and the Advisory Fracture Committee of the American Medical Association. Second edition. St. Louis: The C. V. Mosby Company. 1937. Price $12.50.

"Man, Bread and Destiny. The Story of Man's Food"

By C. C. FURNAS, associate professor of chemical engineering, Yale University, and S. M. FURNAS, formerly instructor in nutrition, University of Minnesota. New York: Reynal & Hitchcock. Price $3.00.

"A Textbook of Exodontia. Exodontia, Oral Surgery and Anesthesia"

By LEO WINTER, M.D., D.D.S., Sc.D. (Hon.), LL.D., professor of oral surgery and director of the Oral and Minor Surgery Clinic, New York University College of Dentistry; visiting dental surgeon in charge, Bellevue Hospital; Lecturer in Oral Surgery, New York Homeopathic Medical School; Oral Surgeon, Flower Hospital; lieutenant commander, D.C.V.(S), N.S.N.R.; special lecturer and instructor in oral surgery, Dental Department, U. S. Naval Medical School; visiting dental surgeon, New York Foundling Hospital. Third revised edition. St. Louis: The C. V. Mosby Company. 1937. Price $10.00.

"Diseases of the Soft Structures of the Teeth and Their Treatment. A Text-Book for Students and Practitioners"

By HERMANN PRINZ, A.M., D.D.S., M.D., D.Sc., Dr. Med. Dent., professor of materia medica and therapeutics, the Thomas W. Evans Museum and Dental Institute School of Dentistry University of Pennsylvania, Philadelphia. Second edition, thoroughly revised. Illustrated with 308 engravings. Philadelphia: Lea & Febiger, 1937. Price $6.50.

"A Text-Book of Medical Bacteriology"

By R. W. FAIRBROTHER, D.Sc., M.D., M.R.C.P., lecturer in bacteriology, University of Manchester; late research fellow in bacteriology, Lister Institute, London. St. Louis: The C. V. Mosby Company. 1937. Price $4.50.

"Physical Diagnosis. The Art and Technique of History Taking and Physical Examination of the Patient in Health and in Disease"

By DON C. SUTTON, M.S., M.D., associate professor of medicine, Northwestern University School of Medicine; attending physician and chairman of the Medical Division of the Cook County Hospital; chief of the Cardiac Clinic, Cook County Hospital, Chicago; attending physician, The Evanston Hospital. With 298 text illustrations, and 8 color plates. St. Louis: The C. V. Mosby Company. 1937. Price $5.00.

"Studies in Dental Prosthetics"

By G. Y. HILDEBRAND, licensed dental surgeon, laborator in prosthetics and orthodontia at the Dental Institute of the Royal Caroline Medico-Chirurgical Institute in Stockholm. Vol. I, text. Vol. II, illustrations. Stockholm: Aktiebolaget Fahlcrantz' Boktryckeri. 1937. Published by the Swedish Dental Association. Supplement to Volume 30.

"The Little Things in Life. The Vitamins, Hormones, and Other Minute Essentials for Health"

By BARNETT SURE, Ph.D., professor of agricultural chemistry in the University of Arkansas. New York and London: D. Appleton-Century Company, Inc. 1937. Price $2.50.

"Take Care of Yourself. A Practical Guide to Health and Beauty. Stressing the Proper Way to Use and the Prudent Way to Buy Home Remedies and Cosmetics"

By JEROME W. EPHRAIM. Foreword by Logan Clendening, M.D., author of "The Human Body." New York: Simon and Schuster, 1937. Price $2.00.

"Synopsis of Digestive Diseases"

By JOHN L. KANTOR, Ph.D., M.D., associate in medicine, Columbia University; gastroenterologist and associate roentgenologist, Montefiore Hospital for Chronic Diseases, New York. Illustrated. St. Louis: The C. V. Mosby Company. 1937. Price $3.50.

"Tissue Reactions in Bone and Dentin. A Morpho-Biological Study of the Formation and the Dissolving of Bone and Dentine"

By AKE WILTON, M.D., associate professor and lecturer in pathology at the Caroline Institute, Stockholm. London: Henry Kimpton. 1937. Price 15s net.

MISCELLANY

NEWS

Denver Summer Seminar for Advanced Study of Orthodontics

The second summer seminar for the study of orthodontics was held from July 17 through July 25 at the University of Colorado School of Medicine in Denver, Colorado. In accordance with the informal plan of the first summer seminar, material was presented relating to orthodontics with reference to the constitutional background of the patient; bone pathology and its orthodontic significance; case analysis; treatment planning; appliance construction; radiography and photography. Perhaps most significant were the impartial discussion and freedom from dogma which characterized both the presentations and discussion groups. It was the expressed wish of those attending that the summer seminar be continued as an annual event and plans were made for a third meeting to be held in the summer of 1938 at a time convenient for those also wishing to attend the meeting of the American Society of Orthodontists.

Deaths

Ammeter, Christian, Portland, Ore.; North Pacific College of Oregon, 1916; died October 6; aged 52.

Anderson, Russell E., Delta, Colo.; School of Dentistry University of Denver, 1926; died August 14; aged 39.

Baxley, C. D., Big Spring, Texas; Baylor University College of Dentistry, 1914; died recently.

Beiser, Joseph W., Philadelphia, Pa.; Temple University School of Dentistry, 1904; died May 24. Dr. Beiser was associate professor of clinical operative dentistry at Temple University.

Black, A. T., Mt. Ayr, Iowa; College of Dentistry, State University of Iowa, 1907; died August 14; aged 54.

Boren, Wilber C., Princeton, Ind.; Indiana University School of Dentistry, 1913; died September 7; aged 53.

Brown, Edward D., Tecumseh, Mich.; University of Michigan College of Dental Surgery, 1896; died October 12.

Cary, Albert E., Hartford, Conn.; Baltimore College of Dental Surgery, 1899; died August 5; aged 61.

Clark, Paul, Knoxville, Iowa; College of Dentistry, State University of Iowa, 1923; died September 21; aged 41.

DeBrot, John C. F., New York City; New York University College of Dentistry, 1906; died September 1.

Demby, Benjamin A., Chicago, Ill.; Dental Department of Meharry Medical College, 1917; died August 2; aged 51.

Drake, J. Francis, Portland, Ore.; North Pacific College of Oregon, 1905; died September 5; aged 68.

Duncan, James, Warsaw, Ind.; Indiana University School of Dentistry, 1907; died August 19; aged 55.

Garretson, Walter R., Knoxville, Iowa; School of Dentistry, University of Pennsylvania, 1895; died September 24; aged 64.

Guest, O. M., Haskell, Texas; died August 28; aged 56.

Hamilton, F. A., Indianapolis, Ind., Indiana University School of Dentistry, 1895; died September 3; aged 65.

Hanson, Oscar W., Madison, S. D., Northwestern University Dental School, 1906; died July 5; aged 56. Dr. Hanson was a past president of the South Dakota State Dental Society.

Hergert, Frank Winfield, Seattle, Wash.; North Pacific College of Oregon, 1903; died September 3; aged 57.

Knapp, Emil, Evansville, Ind., died June 2; aged 83.

Laue, Gilbert F., Gary, Ind.; School of Dentistry University of Pennsylvania, 1900; died August 29; aged 60.

Logan, Gordon Byron, Melcher, Iowa, College of Dentistry, State University of Iowa, 1913; died August 27; aged 50.

Lundgren, Charles J., Brooklyn, N. Y., New York University College of Dentistry, 1890; died September 23.

Machus, Frank, Oconomowoc, Wis., Mar-

quette University Dental School, 1915; died August 25; aged 77.

Moore, Walter P., Marion, Ill.; School of Dentistry, University of Louisville, 1887; died August 8; aged 77.

Nell, Reinhard, Philadelphia, Pa., died June 26. Dr. Nell was a former president of the North Philadelphia Association of Dental Surgeons and at the time of his death a member of the board of governors. He was a tireless and loyal worker in organized dentistry.

Newberger, L. R., Bedford, Ind., Indiana University School of Dentistry, 1918; died August 12.

Noland, Ray W., Des Moines, Ia., College of Dentistry, State University of Iowa, 1917; died October 3; aged 50.

Norfleet, Robert Gordon, Norfolk, Va., Baltimore College of Dental Surgery, 1897; died September 6.

Ross, Frank A., Chicago, Ill., Northwestern University Dental School, 1896; died September 7; aged 72.

Ruen, Michael C., Detroit, Mich., University of Michigan School of Dentistry, 1907; died September 9; aged 64.

Sage, Frank W., Cincinnati, Ohio; Ohio College of Dental Surgery, 1869; died September 25; aged 91.

Sexton, Paul, Waco, Texas; Baylor University College of Dentistry; died August 17.

Seyfert, Robert E., Lebanon, Pa., School of Dentistry, University of Pennsylvania, 1904. Dr. Seyfert died in an automobile accident, September 19.

Smith, George F., Belding, Mich., Temple University School of Dentistry, 1890; died August 29; aged 70.

Stephens, Russell F., Marion, Ind., Indiana University School of Dentistry, 1926; died August 16.

Tennent, Eugene Hunter, Dental Corps, United States Navy. The Navy Dental Corps marks with sorrow the passing of Capt. Eugene Hunter Tennent, one of its most distinguished officers, who died at the U. S. Naval Hospital, Brooklyn, N. Y., October 23 at the age of 54. Captain Tennent, who was among the first group of four officers appointed to the Dental Corps of the Navy, was commissioned as a lieutenant (junior grade) in that Corps on January 11, 1913. He served with merit aboard many naval vessels and at various activities in the United States and abroad. He was one of the first dental officers to serve on a combatant ship, reporting for duty on board the U. S. S. Louisiana November 6, 1914. Wherever stationed, Captain Tennent enjoyed a close contact with the civilian members of the dental profession and was a welcome visitor to society meetings on account of his ability to discuss the various phases of dental practice. He was a member of the American Dental Association, the American College of Dentists and the Association of Military Dental Surgeons.

Van Osdol, L. E., Peru, Ind.; Indiana University School of Dentistry, 1899; died September 7; aged 65.

Webb, G. D., Joliet, Ill. College of Dentistry, State University of Iowa, 1899; died September 17; aged 69.

Webber, S. T. B., Chicago, Ill., Chicago College of Dental Surgery, 1904; died August 14; aged 61.

Webster, Walter J., Jersey City, N. J.; Temple University School of Dentistry, 1905; died October 7; aged 63.

Wetzel, Walter C., Shamokin, Pa., died June 1.

Wofford, C. D., Plainview, Texas; Baltimore College of Dental Surgery, 1912; died recently.

Wyckoff, William T., Philadelphia, Pa., Temple University School of Dentistry, 1887. Dr. Wyckoff, who was one of the early pioneers in the use of local anesthesia, died at his Ocean City home July 31, at the age of 79. Four years ago, he had to have his left leg amputated and a year ago he lost his right leg at the knee, but he continued his practice until his sight was affected. Dr. Wyckoff was a trustee of Temple University Dental School and served in the Dental Corps of the 309th Infantry during the World War. He was an honorary member of the First District Dental Society. Dr. Wyckoff is survived by two sons, both dentists.

BOOKS PUBLISHED IN 1936-1937
which may be borrowed* or purchased† from the LIBRARY BUREAU of the AMERICAN DENTAL ASSOCIATION
212 E. Superior St., Chicago, Ill.

AMERICAN DENTAL ASSN., COUNCIL ON DENTAL THERAPEUTICS: Accepted dental remedies. Chicago: American Dental Assn., 1937. $1.00.

AMERICAN FOUNDATION: American medicine. 2 vols. New York: American Foundation, 1937. $3.50.

BAILEY, HAMILTON: Clinical surgery for dental practitioners. London: Lewis, 1937. $4.00.

BIRCH, C. L.: Hemophilia—clinical and genetic aspects. Urbana, Ill.: Univ. of Illinois, 1937. $2.00 paper, $2.50 cloth.

BLACK, A. D.: G. V. Black's work on operative dentistry with which his special dental pathology is combined. 4 vol. V. 1—Pathology of hard tissues of teeth; oral diagnosis. V. 2—Technical procedures in making restorations in teeth. V. 3—Treatment of dental caries. V. 4—Diseases and treatment of investing tissues of teeth and other soft tissues of mouth. Chicago: Medico-Dental Publishing Co., 1936. Complete set $16.00, $4.50 per volume.

BLAIR, V. P., & IVY, R. H.: Essentials of oral surgery. St. Louis: Mosby, 1936. $6.50.

BLAYNEY, J. R.: Dental pharmacology and therapeutics. St. Louis: Mosby, 1936. $4.00.

BOYLE, H. H.: Principles and practice of applied dental aesthetics. London: Kimpton, 1936. $7.25.

BRODERICK, F. W.: Principles of dental medicine: the medical aspects of dental disease. St. Louis: Mosby, 1936. $7.50.

BUNTING, R. W.: Oral hygiene and the treatment of parodontal diseases. Philadelphia: Lea & Febiger, 1936. $2.50.

CARNEGIE, DALE: How to win friends and influence people. New York: Simon & Schuster, 1937. $1.96.

COOK LABORATORIES: Manual of local anesthesia in general dentistry. New York: Cook Laboratories, Inc., 1936.

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ENNIS, L. M.: Dental roentgenology. Philadelphia: Lea & Febiger, 1936. $6.50.

EPHRAIM, J. W.: Take care of yourself: a practical guide to health and beauty. New York: Simon and Schuster, 1937. $2.00.

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FAIRBROTHER, R. W.: Text-book of medical bacteriology. St. Louis: Mosby, 1937. $4.50.

FALK, I. S.: Security against sickness. New York: Doubleday, Doran, 1936. $4.00.

FISH, E. W.: Principles of full denture prosthesis. London: Bale, 1937. $5.00.

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GREENFIELD, A. L.: X-ray technic and interpretation of dental roentgenograms. Brooklyn: Dental Items Interest, 1936. $6.00.

HADEN, R. L.: Dental infection and systemic disease. Philadelphia: Lea & Febiger, 1936. $2.50.

HILDEBRAND, G. Y.: Studies in dental prosthetics. 2 vols. Stockholm, Sweden: Aktiebolaget Fahlcrantz' Boktryckeri, 1937.

KANTOR, J. L.: Synopsis of digestive diseases. St. Louis: Mosby, 1937. $3.50.

KEY, J. A., & CONWELL, H. E.: Management of fractures, dislocations, and sprains. St. Louis: Mosby, 1937. $12.50.

KRONFELD, RUDOLF: Dental histology and comparative dental anatomy. Philadelphia: Lea & Febiger, 1937. $3.75.

LAMB, RUTH DE F.: American chamber of horrors. New York: Farrar & Rinehart, 1936. $2.50.

LEVINSON, C. A.: Examining dentist in food hazard cases. Boston, Mass.: Author, 1937. $2.00.

MASON, E. G.: Why we do it. Elementary discussion of human conduct and related

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physiology. St. Louis: Mosby, 1937. $1.50.

McBride, W. C.: Juvenile dentistry. Philadelphia: Lea & Febiger, 1937. $6.00.

McClendon, J. F., & Pettibone, C. J. V.: Physiological chemistry. St. Louis: Mosby, 1936. $3.50.

McGehee, W. H. O.: Textbook of dental pharmacology, materia dentica and pharmaco-therapeutics. Philadelphia: Blakiston, 1936. $4.25.

McGehee, W. H. O.: Textbook of operative dentistry. Philadelphia: Blakiston, 1936. $10.00.

Milbank Memorial Fund: Next steps in public health. New York: Milbank Memorial Fund, 1936.

Miller, S. C.: Oral diagnosis and treatment planning. Philadelphia: Blakiston, 1936. $7.50.

Mitchiner, P. H.; Shattock, C. E.; Slesinger, E. G., & Wakeley, C. P. G.: Surgery for dental students. Baltimore: Wood, 1936. $4.75.

Pearlman, L. M.: Your breath and your health. 295 Madison Avenue, New York: Academy Pub. Co., 1936. $1.00.

Prinz, Hermann: Dental formulary. Philadelphia: Lea & Febiger, 1936. $3.50.

Prinz, Hermann: Diseases of soft structures of the teeth and their treatment. Philadelphia: Lea & Febiger, 1937. $6.50.

Rauch, A. W.: Analgesia: nitrous oxide in dentistry. Newark, New Jersey: Author, 1936. $3.50.

Rhinehart, D. A.: Roentgenographic technique. Philadelphia: Lea & Febiger, 1936. $6.50.

Salzmann, J. A.: Principles and practice of public health dentistry. Boston: Stratford, 1937. $4.00.

Schwartz, J. R.: Cavity preparation and abutment construction. Brooklyn: Dental Items Interest, 1936. $6.50.

Shelling, D. H.: Parathyroids in health and in disease. St. Louis: Mosby, 1936. $5.00.

Simkins, C. S.: History of human teeth. Philadelphia: Blakiston, 1937. $4.00.

Skinner, E. W.: Science of dental materials. Philadelphia: Saunders, 1936. $4.50.

Stern, Frances: Applied dietetics. Baltimore: Williams & Wilkins, 1936. $3.50.

Stillman, P. R., & McCall, J. O.: Textbook of clinical periodontia. New York: Macmillan, 1937. $3.50.

Sure, Barnett: Little things in life: vitamins, hormones, and other minute essentials for health. New York: Appleton-Century, 1937. $2.50.

Sutton, D. C.: Physical diagnosis: art and technique of history taking and physical examination of the patient. St. Louis: Mosby, 1937. $5.00.

Thoma, K. H.: Oral diagnosis and treatment planning. Philadelphia: Saunders, 1936. $6.00.

Thompson, W. S.: Operative and interpretive radiodontia. Philadelphia: Lea & Febiger, 1936. $7.00.

Turner, C. E.: Personal hygiene. St. Louis: Mosby, 1937. $2.25.

United States Public Health Service: Dental survey of school children, ages 6-14 years, etc. Public Health Bull. No. 226. Washington, D. C.: Government Printing Office, 1936. 20c.

United States Public Health Service: Survey of dental activities of state departments and institutions of the United States. Public Health Bull. No. 227. Washington, D. C.: Government Printing Office, 1936. 20c.

Wald, S. S.: Manual of dental roentgenology. New York: Guggenheim Dental Clinic, 1936. $2.25.

White, E. Grace: Textbook of general biology. St. Louis: Mosby, 1937. $3.00.

Whiteford, G. H., & Coffin, R. G.: Essentials of college chemistry. St. Louis: Mosby, 1937. $4.00.

Widdowson, T. W.: Dental surgery and pathology. London: Bale, 1937. $9.50.

Wilton, Ake: Tissue reactions in bone and dentine. London: Kimpton, 1937. $4.50.

Winter, Leo: Textbook of exodontia: exodontia, oral surgery and anesthesia. St. Louis: Mosby, 1937. $10.00.

Young, M.; Johnson, E.; Smyth, C., & Still, M.: Investigations into the nature and characteristic features of post-normal occlusion. London: His Majesty's Stationery Office, 1937. $1.25.

Zahorsky, John: Synopsis of pediatrics. St. Louis: Mosby, 1937. $4.00.

Practical English and Effective Speech

By special arrangement with the Better-Speech Institute of America, the Library Bureau is offering, for a limited time, the Institute's self-teaching course in Practical English and Effective Speech at $2.00 prepaid to A.D.A. members. This is the same course that is advertised over the air and the regular retail price is $5.00. The course consists of fifteen 32-page lessons, comprising Vocabulary Development, Grammar, Punctuation, Pronunciation, Spelling, Enunciation and Speech Refinements. Send your order with $2.00 to the Library Bureau.

ANNOUNCEMENTS

ANNOUNCEMENTS must be received by the fifth day of the month in order to be published in the forthcoming issue of THE JOURNAL.

CALENDAR OF MEETINGS

American Dental Association, St. Louis, Mo., October 24-28, 1938.

National Board of Dental Examiners, December 3-4.

American Academy of Restorative Dentistry, St. Louis, Mo., October 22-23, 1938.

American Society for the Advancement of General Anesthesia in Dentistry, New York City, fourth Monday in March and October.

Five State Postgraduate Clinic, Washington, D. C., March 6-9, 1938.

Thomas P. Hinman Midwinter Clinic, Atlanta, Ga., March 14-15, 1938.

District of Columbia Dental Society, second and fourth Tuesdays in each month from October to June at the United States Public Health Service Auditorium, Washington.

Greater New York Dental Meeting, New York City, December 6-10.

Greater Philadelphia Annual Meeting, Philadelphia, February 2-4, 1938.

Dallas Midwinter Dental Clinic, Dallas, Texas, January 17-19, 1938.

North St. Louis District Dental Society, Annual Midwinter Meeting, January 26-27, 1938.

Rocky Mountain Midwinter Meeting, Denver, Colo., January 9-12, 1938.

Harvard Society of Orthodontists, Boston, Mass., January 12, 1938.

Seventh Cruise-Congress of the Pan American Medical Association: Section of Dentistry and Oral Surgery, January 15-31, 1938.

American Dental Society of Europe, Stockholm, August 1-3, 1938.

University of Buffalo School of Dentistry Alumni Association, Buffalo, N. Y., February 23-25, 1938.

International College of Dentists, U. S. Section, Chicago, Ill., February 13, 1938.

STATE SOCIETIES

December

Hawaii, at Honolulu

January

Rhode Island, at Providence (12-13)

February

Minnesota, at St. Paul (22-24)

April

Kansas, at Wichita (24-27)
Wisconsin, at Milwaukee (25-28)

May

Missouri, at Columbia (8-9)
New York, at Syracuse (10-13)
North Carolina, at Winston-Salem (2-4)
Pennsylvania, at Philadelphia (3-5)
South Dakota, at Watertown (23-24)
Tennessee, at Chattanooga (9-12)

June

Colorado, at Estes Park (20-22)

STATE BOARDS OF DENTAL EXAMINERS

California, at San Francisco, December 6. Kenneth I. Nesbitt, 450 McAllister St., San Francisco, Secretary.

Connecticut, at Hartford, November 30-December 4. Almond J. Cutting, Southington, Recorder.

Louisiana, at New Orleans, December 16-18. Joseph L. Webre, 740 Audubon Bldg., Secretary.

Minnesota, at Minneapolis, December 10-16. Paul Hagen, Crookston, Secretary.

New Jersey, December 6-11. Walter A. Wilson, 148 W. State St., Trenton, Secretary.

Texas, at Houston, December 13-16. B. Carl Holder, 1009 Nixon Bldg., Corpus Christi, Secretary.

Wisconsin, at Milwaukee, December 13-17. S. F. Donovan, Tomah, Secretary.

COLORADO STATE DENTAL ASSOCIATION

The fifty-second annual meeting of the Colorado State Dental Association will be

held at the Stanley Hotel, Estes Park, June 20-22, 1938.

RALPH L. CHRISTY, *Secretary,*
820 Majestic Bldg.,
Denver.

KANSAS STATE DENTAL ASSOCIATION

The sixty-seventh annual session of the Kansas State Dental Association will be held in the Allis Hotel, Wichita, April 24-27, 1938.

FRED A. RICHMOND, *Secretary,*
1008 Huron Bldg.,
Kansas City.

DENTAL SOCIETY OF THE STATE OF NEW YORK

The seventieth annual meeting of the Dental Society of the State of New York will be held May 10-13, 1938, at the Hotel Syracuse, Syracuse.

CARA CARELLI, *Executive Secretary,*
1 Hanson Place,
Brooklyn.

RHODE ISLAND STATE DENTAL SOCIETY

The annual meeting of the Rhode Island State Dental Society will be held January 12-13 at the Biltmore Hotel, Providence.

JAMES C. KRASNOFF, *Chairman,*
Clinic Committee.

PENNSYLVANIA STATE DENTAL SOCIETY

The seventieth annual meeting of the Pennsylvania State Dental Society will be held at the Benjamin Franklin Hotel, Philadelphia, May 3-5, 1938.

C. J. HOLLISTER, *Business Manager,*
800 Capital St.,
Harrisburg.

THOMAS P. HINMAN MIDWINTER CLINIC

The twenty-fourth annual session of the Thomas P. Hinman Midwinter Clinic will be held at the Biltmore Hotel, Atlanta, Ga., March 14-15, 1938.

J. D. OSBORNE, *Secretary,*
Doctors' Bldg.,
Atlanta, Ga.

PAN AMERICAN MEDICAL ASSOCIATION: SECTION OF DENTISTRY AND ORAL SURGERY

The Section of Dentistry and Oral Surgery of the Pan American Medical Association will participate in the Seventh Cruise-Congress. In addition to the social activities, there will be papers and clinics by dentists and physicians, with general assemblies and round table discussions aboard ship. Physicians and dentists resident in the countries visited will be entertained at luncheons aboard ship. The Cruise-Congress boat, "Queen of Bermuda," sails from New York January 15 and returns to port January 31, spending five days in Havana and one day each in Port au Prince, Trujillo City and San Juan.

WILLIAM H. PARKE, *Secretary,*
630 Fifth Ave.,
New York City.

INTERNATIONAL COLLEGE OF DENTISTRY

The next annual meeting of the International College of Dentists, United States Section, will be held at the Stevens Hotel, Chicago, Ill., February 13, 1938.

ELMER S. BEST, *Registrar,*
801 Medical Arts Bldg.,
Minneapolis, Minn.

HARVARD SOCIETY OF ORTHODONTISTS

The semi-annual meeting and dinner of the Harvard Society of Orthodontists will be held at Vanderbilt Hall, 107 Louis Pasteur Ave., Boston, Mass., January 12, 1938, at 6:30 o'clock.

HAROLD J. NICE, *Secretary,*
475 Commonwealth Ave.,
Boston, Mass.

UNIVERSITY OF BUFFALO SCHOOL OF DENTISTRY ALUMNI ASSOCIATION

The University of Buffalo School of Dentistry Alumni Association will hold its thirty-eighth annual meeting in Buffalo, N. Y., at the Hotel Statler, February 23-25, 1938.

TRACY M. BISSELL, *Chairman,*
Publicity Committee.

DINNER IN HONOR OF DR. POSNER

The dental staff of St. Luke's Hospital of New York City will give a dinner Saturday evening, December 4, at the Continental Ballroom of the Hotel St. Moritz, New York City, in honor of John Jacob Posner on his return from Spain. All members of the dental profession are invited; tickets, $3.50. Address requests for reservations, with check, to

JEROME M. SCHWEITZER,
730 Fifth Ave.,
New York City.

DALLAS MIDWINTER DENTAL CLINIC

The eleventh annual Dallas Midwinter Dental Clinic, sponsored by the Dallas County Dental Society, will be held at the Hotel Adolphus, Dallas, Texas, January 17-19. The study courses are open to all members of the American Dental Association and the fee of $15 includes a ticket to the banquet, at which time the table clinics will be presented. For information, address T. A. Lipscomb, Medical Arts Bldg., Dallas. For reservations for the golf tournament Sunday, January 16, address Judd M. McMinn, Medical Arts Bldg.

P. J. MURPHEY, *Publicity Chairman,*
Northeast Corner Fairmount
and Welborn, Dallas.

UNITED STATES PATENTS APPLICABLE OR PERTAINING TO DENTISTRY ISSUED DURING OCTOBER 1937

October 5

No. 2,094,671, to PAUL POETSCHKE. Composition of Matter Suitable for a Dentifrice.

No. 2,094,888, to WILLIAM J. HOOPER. Dental Cleaning Spray.

No. 2,094,903, to RALPH A. PITTMAN. Dental Model Articulator.

No. 2,095,154, to JAMES SCOTT. Sterilizer.

October 12

No. 2,095,452, to GEORGE P. BRENNER. Denture.

No. 2,095,511, to WILLIAM T. OBERTO. Anesthesia Demonstration Device.

No. 2,095,535, to MERRILL G. SWENSON. Artificial Tooth.

No. 2,095,543, to PETER DOLL. Container for Dental Amalgams.

No. 2,095,581, to BYRON E. WHITE. Mechanical Toothbrush.

No. 2,095,665, to AUGUST S. GRETH. Paralleling and Surveying Machine.

No. 2,095,741, to ELMER E. HACHMAN. Toothbrush.

October 19

No. 2,096,375, to WILLIAM B. JONES. Dentist Apparatus.

No. 2,096,515, to CARL HILTEBRANDT. Dental Bridge.

October 26

No. 2,097,010, to ALFRED ARNEGGER. Toothbrush.

No. 2,097,047, to LAKE H. SPRINKLE. Artificial Tooth and Means for Connecting the Same to Dental Plates or Dentures.

No. 2,097,098, to NORMAN L. HISCOX. Electric Spatula.

No. 2,097,167, to BERT B. SWINNERTON. Dental Lavatory.

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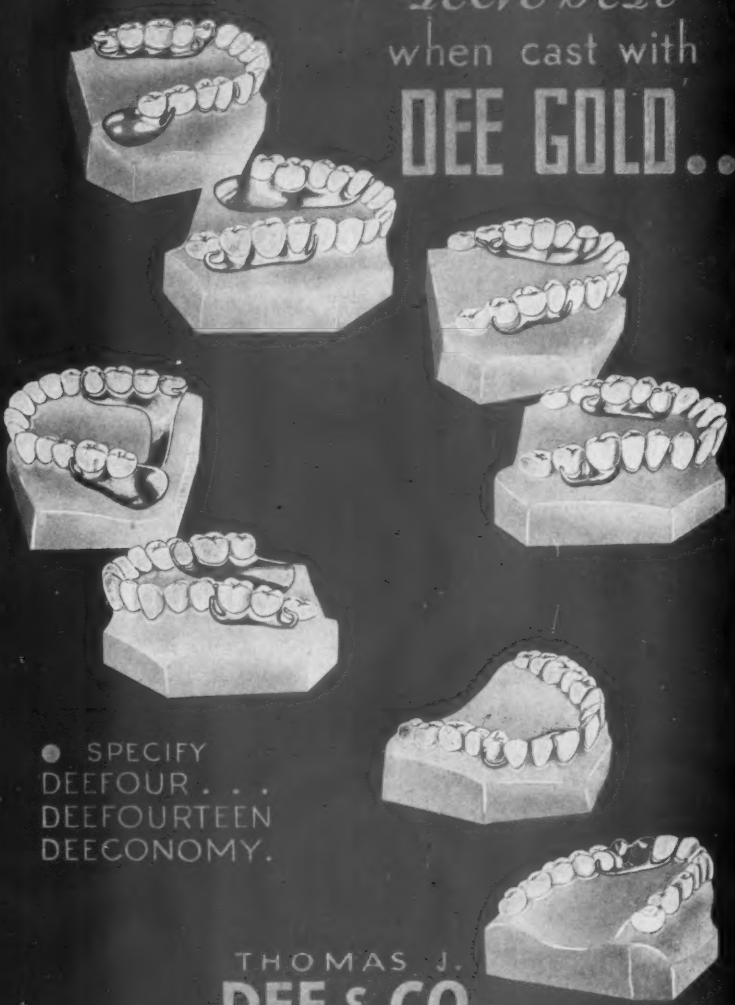

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